Specifiers

For sexually mature individuals, the following specifiers may be noted based on the individual's sexual orientation: **Sexually Attracted to Males, Sexually Attracted to Females, Sexually Attracted to Both,** and **Sexually Attracted to Neither.** Males with Gender Identity Disorder include substantial proportions with all four specifiers. Those who are attracted to males usually first experience the disorder beginning in childhood or early adolescence, while those males attracted to females, both genders, or neither usually report their gender dysphoria beginning in early to mid-adulthood. Those men attracted to neither gender are often isolated individuals with schizoid traits. Virtually all females with Gender Identity Disorder will receive the same specifier—Sexually Attracted to Females—although there are exceptional cases involving females who are Sexually Attracted to Males.

Recording Procedures

The assigned diagnostic code depends on the individual's current age: if the disorder occurs in childhood, the code 302.6 is used; for an adolescent or adult, 302.85 is used.

Associated Features and Disorders

Associated descriptive features and mental disorders. Many individuals with Gender Identity Disorder become socially isolated. Isolation and ostracism contribute to low self-esteem and may lead to school aversion or dropping out of school. Peer ostracism and teasing are especially common sequelae for boys with the disorder. Boys with Gender Identity Disorder often show marked feminine mannerisms and speech patterns.

The disturbance can be so pervasive that the mental lives of some individuals revolve only around those activities that lessen gender distress. They are often preoccupied with appearance, especially early in the transition to living in the opposite sex role. Relationships with one or both parents also may be seriously impaired. Some males with Gender Identity Disorder resort to self-treatment with hormones and may very rarely perform their own castration or penectomy. Especially in urban centers, some males with the disorder may engage in prostitution, which places them at high risk for human immunodeficiency virus (HIV) infection. Suicide attempts and Substance-Related Disorders are commonly associated.

Children with Gender Identity Disorder may manifest coexisting Separation Anxiety Disorder, Generalized Anxiety Disorder, and symptoms of depression. Adolescents are particularly at risk for depression and suicidal ideation and suicide attempts. In adults, anxiety and depressive symptoms may be present. In clinical samples, associated Personality Disorders are more common among males than among females. Adult males who are sexually attracted to females, to both males and females, or to neither sex usually report a history of erotic arousal associated with the thought or image of oneself as a woman (termed *autogynephilia*). In most cases, the individual would qualify, at least in his past, for a diagnosis of Transvestic Fetishism. In others, however, the individual's favorite fantasy emphasizes feminine attributes other than clothing. Some men, for example, masturbate while picturing themselves

as nude women, focusing on their imagined breasts and vulvas; others masturbate while picturing themselves engaged in some stereotypically feminine activity such as knitting.

Associated laboratory findings. There is no diagnostic test specific for Gender Identity Disorder. In the presence of a normal physical examination, karyotyping for sex chromosomes and sex hormone assays are usually not indicated. Psychological testing may reveal cross-gender identification or behavior patterns.

Associated physical examination findings and general medical conditions. Individuals with Gender Identity Disorder have normal genitalia (in contrast to the ambiguous genitalia or hypogonadism found in physical intersex conditions). Adolescent and adult males with Gender Identity Disorder may show breast enlargement resulting from hormone ingestion, hair denuding from temporary or permanent epilation, and other physical changes as a result of procedures such as rhinoplasty or thyroid cartilage shaving (surgical reduction of the Adam's apple). Distorted breasts or breast rashes may be seen in females who wear breast binders. Postsurgical complications in genetic females include prominent chest wall scars, and in genetic males, vaginal strictures, rectovaginal fistulas, urethral stenoses, and misdirected urinary streams. Adult females with Gender Identity Disorder may have a higher-than-expected likelihood of polycystic ovarian disease.

Specific Age and Gender Features

Females with Gender Identity Disorders generally experience less ostracism because of cross-gender interests and may suffer less from peer rejection, at least until adolescence. In child clinic samples, boys with this disorder are referred for evaluation much more frequently than are girls. In adult clinic samples, men outnumber women by about two or three times. In children, the referral bias toward males may partly reflect the greater stigma that cross-gender behavior carries for boys than for girls.

Prevalence

There are no recent epidemiological studies to provide data on prevalence of Gender Identity Disorder. Data from smaller countries in Europe with access to total population statistics and referrals suggest that roughly 1 per 30,000 adult males and 1 per 100,000 adult females seek sex-reassignment surgery.

Course

For clinically referred children, onset of cross-gender interests and activities is usually between ages 2 and 4 years, and some parents report that their child has always had cross-gender interests. Only a very small number of children with Gender Identity Disorder will continue to have symptoms that meet criteria for Gender Identity Disorder in adolescence or adulthood. Typically, children are referred around the time of school entry because of parental concern that what they regarded as a "phase" does not appear to be passing. Most children with Gender Identity Disorder display

less overt cross-gender behaviors with time, parental intervention, or response from peers. By late adolescence or adulthood, about three-quarters of boys who had a childhood history of Gender Identity Disorder report a homosexual or bisexual orientation, but without concurrent Gender Identity Disorder. Most of the remainder report a heterosexual orientation, also without concurrent Gender Identity Disorder. The corresponding percentages for sexual orientation in girls are not known. Some adolescents may develop a clearer cross-gender identification and request sex-reassignment surgery or may continue in a chronic course of gender confusion or dysphoria.

In adult males, there are two different courses for the development of Gender Identity Disorder. The first is a continuation of Gender Identity Disorder that had an onset in childhood. These individuals typically present in late adolescence or adulthood. In the other course, the more overt signs of cross-gender identification appear later and more gradually, with a clinical presentation in early to mid-adulthood usually following, but sometimes concurrent with, Transvestic Fetishism. The later-onset group may be more fluctuating in the degree of cross-gender identification, more ambivalent about sex-reassignment surgery, more likely to be sexually attracted to women, and less likely to be satisfied after sex-reassignment surgery. Males with Gender Identity Disorder who are sexually attracted to males tend to present in adolescence or early adulthood with a lifelong history of gender dysphoria. In contrast, those who are sexually attracted to females, to both males and females, or to neither sex tend to present later and typically have a history of Transvestic Fetishism. Typically, after sex reassignment, those males who were attracted to females wish to live with another woman in either a lesbian relationship or as sisters. If Gender Identity Disorder is present in adulthood, it tends to have a chronic course, but spontaneous remission has been reported.

Differential Diagnosis

Gender Identity Disorder can be distinguished from simple **nonconformity to stereotypical sex-role behavior** by the extent and pervasiveness of the cross-gender wishes, interests, and activities. This disorder is not meant to describe a child's nonconformity to stereotypic sex-role behavior as, for example, in "tomboyishness" in girls or "sissyish" behavior in boys. Rather, it represents a profound disturbance of the individual's sense of identity with regard to maleness or femaleness. Behavior in children that merely does not fit the cultural stereotype of masculinity or femininity should not be given the diagnosis unless the full syndrome is present, including marked distress or impairment.

Transvestic Fetishism occurs in heterosexual (or bisexual) men for whom the cross-dressing behavior is for the purpose of sexual excitement. Aside from cross-dressing, most individuals with Transvestic Fetishism do not have a history of childhood cross-gender behaviors. Males with a presentation that meets full criteria for Gender Identity Disorder as well as Transvestic Fetishism should be given both diagnoses. If gender dysphoria is present in an individual with Transvestic Fetishism but full criteria for Gender Identity Disorder are not met, the specifier With Gender Dysphoria can be used.

The category **Gender Identity Disorder Not Otherwise Specified** can be used for

individuals who have a gender identity problem with a **concurrent congenital intersex condition** (e.g., partial androgen insensitivity syndrome or congenital adrenal hyperplasia).

In **Schizophrenia,** there may rarely be delusions of belonging to the other sex. Insistence by a person with a Gender Identity Disorder that he or she is of the other sex is not considered a delusion, because what is invariably meant is that the person feels like a member of the other sex rather than truly believes that he or she is a member of the other sex. In very rare cases, however, Schizophrenia and severe Gender Identity Disorder may coexist.

Diagnostic criteria for Gender Identity Disorder

A. A strong and persistent cross-gender identification (not merely a desire for any perceived cultural advantages of being the other sex).

In children, the disturbance is manifested by four (or more) of the following:

(1) repeatedly stated desire to be, or insistence that he or she is, the other sex
(2) in boys, preference for cross-dressing or simulating female attire; in girls, insistence on wearing only stereotypical masculine clothing
(3) strong and persistent preferences for cross-sex roles in make-believe play or persistent fantasies of being the other sex
(4) intense desire to participate in the stereotypical games and pastimes of the other sex
(5) strong preference for playmates of the other sex

In adolescents and adults, the disturbance is manifested by symptoms such as a stated desire to be the other sex, frequent passing as the other sex, desire to live or be treated as the other sex, or the conviction that he or she has the typical feelings and reactions of the other sex.

B. Persistent discomfort with his or her sex or sense of inappropriateness in the gender role of that sex.

In children, the disturbance is manifested by any of the following: in boys, assertion that his penis or testes are disgusting or will disappear or assertion that it would be better not to have a penis, or aversion toward rough-and-tumble play and rejection of male stereotypical toys, games, and activities; in girls, rejection of urinating in a sitting position, assertion that she has or will grow a penis, or assertion that she does not want to grow breasts or menstruate, or marked aversion toward normative feminine clothing.

In adolescents and adults, the disturbance is manifested by symptoms such as preoccupation with getting rid of primary and secondary sex characteristics (e.g., request for hormones, surgery, or other procedures to physically alter sexual characteristics to simulate the other sex) or belief that he or she was born the wrong sex.

C. The disturbance is not concurrent with a physical intersex condition.

D. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Diagnostic criteria for Gender Identity Disorder *(continued)*

Code based on current age:

302.6 Gender Identity Disorder in Children
302.85 Gender Identity Disorder in Adolescents or Adults

Specify if (for sexually mature individuals):

Sexually Attracted to Males
Sexually Attracted to Females
Sexually Attracted to Both
Sexually Attracted to Neither

302.6 Gender Identity Disorder Not Otherwise Specified

This category is included for coding disorders in gender identity that are not classifiable as a specific Gender Identity Disorder. Examples include

1. Intersex conditions (e.g., partial androgen insensitivity syndrome or congenital adrenal hyperplasia) and accompanying gender dysphoria
2. Transient, stress-related cross-dressing behavior
3. Persistent preoccupation with castration or penectomy without a desire to acquire the sex characteristics of the other sex

302.9 Sexual Disorder Not Otherwise Specified

This category is included for coding a sexual disturbance that does not meet the criteria for any specific Sexual Disorder and is neither a Sexual Dysfunction nor a Paraphilia. Examples include

1. Marked feelings of inadequacy concerning sexual performance or other traits related to self-imposed standards of masculinity or femininity
2. Distress about a pattern of repeated sexual relationships involving a succession of lovers who are experienced by the individual only as things to be used
3. Persistent and marked distress about sexual orientation

Eating Disorders

The Eating Disorders are characterized by severe disturbances in eating behavior. This section includes two specific diagnoses, Anorexia Nervosa and Bulimia Nervosa. **Anorexia Nervosa** is characterized by a refusal to maintain a minimally normal body weight. **Bulimia Nervosa** is characterized by repeated episodes of binge eating followed by inappropriate compensatory behaviors such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise. A disturbance in perception of body shape and weight is an essential feature of both Anorexia Nervosa and Bulimia Nervosa. An Eating Disorder Not Otherwise Specified category is also provided for coding disorders that do not meet criteria for a specific Eating Disorder.

Simple obesity is included in the *International Classification of Diseases* (ICD) as a general medical condition but does not appear in DSM-IV because it has not been established that it is consistently associated with a psychological or behavioral syndrome. However, when there is evidence that psychological factors are of importance in the etiology or course of a particular case of obesity, this can be indicated by noting the presence of Psychological Factors Affecting Medical Condition (p. 731).

Disorders of Feeding and Eating that are usually first diagnosed in infancy or early childhood (i.e., Pica, Rumination Disorder, and Feeding Disorder of Infancy or Early Childhood) are included in the section "Feeding and Eating Disorders of Infancy or Early Childhood" (p. 103).

307.1 Anorexia Nervosa

Diagnostic Features

The essential features of Anorexia Nervosa are that the individual refuses to maintain a minimally normal body weight, is intensely afraid of gaining weight, and exhibits a significant disturbance in the perception of the shape or size of his or her body. In addition, postmenarcheal females with this disorder are amenorrheic. (The term *anorexia* is a misnomer because loss of appetite is rare.)

The individual maintains a body weight that is below a minimally normal level for age and height (Criterion A). When Anorexia Nervosa develops in an individual during childhood or early adolescence, there may be failure to make expected weight gains (i.e., while growing in height) instead of weight loss.

Criterion A provides a guideline for determining when the individual meets the threshold for being underweight. It suggests that the individual weigh less than 85% of that weight that is considered normal for that person's age and height (usually

computed using one of several published versions of the Metropolitan Life Insurance tables or pediatric growth charts). An alternative and somewhat stricter guideline (used in the ICD-10 Diagnostic Criteria for Research) requires that the individual have a body mass index (BMI) (calculated as weight in kilograms/height in meters^2) equal to or below 17.5 kg/m^2. These cutoffs are provided only as suggested guidelines for the clinician, since it is unreasonable to specify a single standard for minimally normal weight that applies to all individuals of a given age and height. In determining a minimally normal weight, the clinician should consider not only such guidelines but also the individual's body build and weight history.

Usually weight loss is accomplished primarily through reduction in total food intake. Although individuals may begin by excluding from their diet what they perceive to be highly caloric foods, most eventually end up with a very restricted diet that is sometimes limited to only a few foods. Additional methods of weight loss include purging (i.e., self-induced vomiting or the misuse of laxatives or diuretics) and increased or excessive exercise.

Individuals with this disorder intensely fear gaining weight or becoming fat (Criterion B). This intense fear of becoming fat is usually not alleviated by the weight loss. In fact, concern about weight gain often increases even as actual weight continues to decrease.

The experience and significance of body weight and shape are distorted in these individuals (Criterion C). Some individuals feel globally overweight. Others realize that they are thin but are still concerned that certain parts of their bodies, particularly the abdomen, buttocks, and thighs, are "too fat." They may employ a wide variety of techniques to estimate their body size or weight, including excessive weighing, obsessive measuring of body parts, and persistently using a mirror to check for perceived areas of "fat." The self-esteem of individuals with Anorexia Nervosa is highly dependent on their body shape and weight. Weight loss is viewed as an impressive achievement and a sign of extraordinary self-discipline, whereas weight gain is perceived as an unacceptable failure of self-control. Though some individuals with this disorder may acknowledge being thin, they typically deny the serious medical implications of their malnourished state.

In postmenarcheal females, amenorrhea (due to abnormally low levels of estrogen secretion that are due in turn to diminished pituitary secretion of follicle-stimulating hormone [FSH] and luteinizing hormone [LH]) is an indicator of physiological dysfunction in Anorexia Nervosa (Criterion D). Amenorrhea is usually a consequence of the weight loss but, in a minority of individuals, may actually precede it. In prepubertal females, menarche may be delayed by the illness.

The individual is often brought to professional attention by family members after marked weight loss (or failure to make expected weight gains) has occurred. If individuals seek help on their own, it is usually because of their subjective distress over the somatic and psychological sequelae of starvation. It is rare for an individual with Anorexia Nervosa to complain of weight loss per se. Individuals with Anorexia Nervosa frequently lack insight into, or have considerable denial of, the problem and may be unreliable historians. It is therefore often necessary to obtain information from parents or other outside sources to evaluate the degree of weight loss and other features of the illness.

Subtypes

The following subtypes can be used to specify the presence or absence of regular binge eating or purging during the current episode of Anorexia Nervosa:

Restricting Type. This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, or excessive exercise. During the current episode, these individuals have not regularly engaged in binge eating or purging.

Binge-Eating/Purging Type. This subtype is used when the individual has regularly engaged in binge eating or purging (or both) during the current episode. Most individuals with Anorexia Nervosa who binge eat also purge through self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Some individuals included in this subtype do not binge eat, but do regularly purge after the consumption of small amounts of food. It appears that most individuals with Binge-Eating/Purging Type engage in these behaviors at least weekly, but sufficient information is not available to justify the specification of a minimum frequency.

Associated Features and Disorders

Associated descriptive features and mental disorders. When seriously underweight, many individuals with Anorexia Nervosa manifest depressive symptoms such as depressed mood, social withdrawal, irritability, insomnia, and diminished interest in sex. Such individuals may have symptomatic presentations that meet criteria for Major Depressive Disorder. Because these features are also observed in individuals without Anorexia Nervosa who are undergoing starvation, many of the depressive features may be secondary to the physiological sequelae of semistarvation. Symptoms of mood disturbance must therefore be reassessed after partial or complete weight restoration.

Obsessive-compulsive features, both related and unrelated to food, are often prominent. Most individuals with Anorexia Nervosa are preoccupied with thoughts of food. Some collect recipes or hoard food. Observations of behaviors associated with other forms of starvation suggest that obsessions and compulsions related to food may be caused or exacerbated by undernutrition. When individuals with Anorexia Nervosa exhibit obsessions and compulsions that are not related to food, body shape, or weight, an additional diagnosis of Obsessive-Compulsive Disorder may be warranted.

Other features sometimes associated with Anorexia Nervosa include concerns about eating in public, feelings of ineffectiveness, a strong need to control one's environment, inflexible thinking, limited social spontaneity, perfectionism, and overly restrained initiative and emotional expression. A substantial portion of individuals with Anorexia Nervosa have a personality disturbance that meets criteria for at least one Personality Disorder. Compared with individuals with Anorexia Nervosa, Restricting Type, those with the Binge-Eating/Purging Type are more likely to have other impulse-control problems, to abuse alcohol or other drugs, to exhibit more mood lability, to be sexually active, to have a greater frequency of suicide attempts in

their history, and to have a personality disturbance that meets criteria for Borderline Personality Disorder.

Associated laboratory findings. Although some individuals with Anorexia Nervosa exhibit no laboratory abnormalities, the semistarvation characteristic of this disorder can affect most major organ systems and produce a variety of disturbances. The induced vomiting and abuse of laxatives, diuretics, and enemas can also cause a number of disturbances leading to abnormal laboratory findings.

Hematology: Leukopenia and mild anemia are common; thrombocytopenia occurs rarely.

Chemistry: Dehydration may be reflected by an elevated blood urea nitrogen (BUN). Hypercholesterolemia is common. Liver function tests may be elevated. Hypomagnesemia, hypozincemia, hypophosphatemia, and hyperamylasemia are occasionally found. Induced vomiting may lead to metabolic alkalosis (elevated serum bicarbonate), hypochloremia, and hypokalemia, and laxative abuse may cause a metabolic acidosis. Serum thyroxine (T_4) levels are usually in the low-normal range; triiodothyronine (T_3) levels are decreased. Hyperadrenocorticism and abnormal responsiveness to a variety of neuroendocrine challenges are common.

In females, low serum estrogen levels are present, whereas males have low levels of serum testosterone. There is a regression of the hypothalamic-pituitary-gonadal axis in both sexes in that the 24-hour pattern of secretion of luteinizing hormone (LH) resembles that normally seen in prepubertal or pubertal individuals.

Electrocardiography: Sinus bradycardia and, rarely, arrhythmias are observed.

Electroencephalography: Diffuse abnormalities, reflecting a metabolic encephalopathy, may result from significant fluid and electrolyte disturbances.

Brain imaging: An increase in the ventricular-brain ratio secondary to starvation is often seen.

Resting energy expenditure: This is often significantly reduced.

Associated physical examination findings and general medical conditions. Many of the physical signs and symptoms of Anorexia Nervosa are attributable to starvation. In addition to amenorrhea, there may be complaints of constipation, abdominal pain, cold intolerance, lethargy, and excess energy. The most obvious finding on physical examination is emaciation. There may also be significant hypotension, hypothermia, and dryness of skin. Some individuals develop lanugo, a fine downy body hair, on their trunks. Most individuals with Anorexia Nervosa exhibit bradycardia. Some develop peripheral edema, especially during weight restoration or on cessation of laxative and diuretic abuse. Rarely, petechiae, usually on the extremities, may indicate a bleeding diathesis. Some individuals evidence a yellowing of the skin associated with hypercarotenemia. Hypertrophy of the salivary glands, particularly the parotid glands, may be present. Individuals who induce vomiting may have dental enamel erosion and some may have scars or calluses on the dorsum of the hand from contact with the teeth when using the hand to induce vomiting.

The semistarvation of Anorexia Nervosa, and the purging behaviors sometimes associated with it, can result in significant associated general medical conditions. These include the development of normochromic normocytic anemia, impaired renal function (associated with chronic dehydration and hypokalemia), cardiovascular

problems (severe hypotension, arrhythmias), dental problems, and osteoporosis (resulting from low calcium intake and absorption, reduced estrogen secretion, and increased cortisol secretion).

Specific Culture, Age, and Gender Features

Anorexia Nervosa appears to be far more prevalent in industrialized societies, in which there is an abundance of food and in which, especially for females, being considered attractive is linked to being thin. The disorder is probably most common in the United States, Canada, Europe, Australia, Japan, New Zealand, and South Africa, but little systematic work has examined prevalence in other cultures. Immigrants from cultures in which the disorder is rare who emigrate to cultures in which the disorder is more prevalent may develop Anorexia Nervosa as thin-body ideals are assimilated. Cultural factors may also influence the manifestations of the disorder. For example, in some cultures, disturbed perception of the body or fear of weight gain may not be prominent and the expressed motivation for food restriction may have a different content, such as epigastric discomfort or distaste for food.

Anorexia Nervosa rarely begins before puberty, but there are suggestions that the severity of associated mental disturbances may be greater among prepubertal individuals who develop the illness. However, data also suggest that when the illness begins during early adolescence (between ages 13 and 18 years), it may be associated with a better prognosis. More than 90% of cases of Anorexia Nervosa occur in females.

Prevalence

The lifetime prevalence of Anorexia Nervosa among females is approximately 0.5%. Individuals who are subthreshold for the disorder (i.e., with Eating Disorder Not Otherwise Specified) are more commonly encountered. The prevalence of Anorexia Nervosa among males is approximately one-tenth that among females. The incidence of Anorexia Nervosa appears to have increased in recent decades.

Course

Anorexia Nervosa typically begins in mid- to late adolescence (age 14–18 years). The onset of this disorder rarely occurs in females over age 40 years. The onset of illness may be associated with a stressful life event. The course and outcome of Anorexia Nervosa are highly variable. Some individuals with Anorexia Nervosa recover fully after a single episode, some exhibit a fluctuating pattern of weight gain followed by relapse, and others experience a chronically deteriorating course of the illness over many years. With time, particularly within the first 5 years of onset, a significant fraction of individuals with the Restricting Type of Anorexia Nervosa develop binge eating, indicating a change to the Binge Eating/Purging subtype. A sustained shift in clinical presentation (e.g., weight gain plus the presence of binge eating and purging) may eventually warrant a change in diagnosis to Bulimia Nervosa.

Hospitalization may be required to restore weight and to address fluid and electrolyte imbalances. Of individuals admitted to university hospitals, the long-term

mortality from Anorexia Nervosa is over 10%. Death most commonly results from starvation, suicide, or electrolyte imbalance.

Familial Pattern

There is an increased risk of Anorexia Nervosa among first-degree biological relatives of individuals with the disorder. An increased risk of Mood Disorders has also been found among first-degree biological relatives of individuals with Anorexia Nervosa, particularly relatives of individuals with the Binge-Eating/Purging Type. Studies of Anorexia Nervosa in twins have found concordance rates for monozygotic twins to be significantly higher than those for dizygotic twins.

Differential Diagnosis

Other possible causes of significant weight loss should be considered in the differential diagnosis of Anorexia Nervosa, especially when the presenting features are atypical (such as an onset of illness after age 40 years). In **general medical conditions** (e.g., gastrointestinal disease, brain tumors, occult malignancies, and acquired immunodeficiency syndrome [AIDS]), serious weight loss may occur, but individuals with such disorders usually do not have a distorted body image and a desire for further weight loss. The **superior mesenteric artery syndrome** (characterized by postprandial vomiting secondary to intermittent gastric outlet obstruction) should be distinguished from Anorexia Nervosa, although this syndrome may sometimes develop in individuals with Anorexia Nervosa because of their emaciation. In **Major Depressive Disorder,** severe weight loss may occur, but most individuals with Major Depressive Disorder do not have a desire for excessive weight loss or excessive fear of gaining weight. In **Schizophrenia,** individuals may exhibit odd eating behavior and occasionally experience significant weight loss, but they rarely show the fear of gaining weight and the body image disturbance required for a diagnosis of Anorexia Nervosa.

Some of the features of Anorexia Nervosa are part of the criteria sets for **Social Phobia, Obsessive-Compulsive Disorder,** and **Body Dysmorphic Disorder.** Specifically, individuals may be humiliated or embarrassed to be seen eating in public, as in Social Phobia; may exhibit obsessions and compulsions related to food, as in Obsessive-Compulsive Disorder; or may be preoccupied with an imagined defect in bodily appearance, as in Body Dysmorphic Disorder. If the individual with Anorexia Nervosa has social fears that are limited to eating behavior alone, the diagnosis of Social Phobia should not be made, but social fears unrelated to eating behavior (e.g., excessive fear of speaking in public) may warrant an additional diagnosis of Social Phobia. Similarly, an additional diagnosis of Obsessive-Compulsive Disorder should be considered only if the individual exhibits obsessions and compulsions unrelated to food (e.g., an excessive fear of contamination), and an additional diagnosis of Body Dysmorphic Disorder should be considered only if the distortion is unrelated to body shape and size (e.g., preoccupation that one's nose is too big).

In **Bulimia Nervosa,** individuals exhibit recurrent episodes of binge eating, engage in inappropriate behavior to avoid weight gain (e.g., self-induced vomiting), and are overly concerned with body shape and weight. However, unlike individuals with

Anorexia Nervosa, Binge-Eating/Purging Type, individuals with Bulimia Nervosa are able to maintain body weight at or above a minimally normal level.

Diagnostic criteria for 307.1 Anorexia Nervosa

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.

D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)

Specify type:

Restricting Type: during the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)

Binge-Eating/Purging Type: during the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)

307.51 Bulimia Nervosa

Diagnostic Features

The essential features of Bulimia Nervosa are binge eating and inappropriate compensatory methods to prevent weight gain. In addition, the self-evaluation of individuals with Bulimia Nervosa is excessively influenced by body shape and weight. To qualify for the diagnosis, the binge eating and the inappropriate compensatory behaviors must occur, on average, at least twice a week for 3 months (Criterion C).

A *binge* is defined as eating in a discrete period of time an amount of food that is definitely larger than most individuals would eat under similar circumstances (Criterion A1). The clinician should consider the context in which the eating occurred—what would be regarded as excessive consumption at a typical meal might be considered normal during a celebration or holiday meal. A "discrete period of time" refers to a limited period, usually less than 2 hours. A single episode of binge eating need not be restricted to one setting. For example, an individual may begin a binge in a restaurant and then continue it on returning home. Continual snacking on small amounts of food throughout the day would not be considered a binge.

Although the type of food consumed during binges varies, it typically includes

sweet, high-calorie foods such as ice cream or cake. However, binge eating appears to be characterized more by an abnormality in the amount of food consumed than by a craving for a specific nutrient, such as carbohydrate. Although individuals with Bulimia Nervosa consume more calories during an episode of binge eating than persons without Bulimia Nervosa consume during a meal, the fractions of calories derived from protein, fat, and carbohydrate are similar.

Individuals with Bulimia Nervosa are typically ashamed of their eating problems and attempt to conceal their symptoms. Binge eating usually occurs in secrecy, or as inconspicuously as possible. An episode may or may not be planned in advance and is usually (but not always) characterized by rapid consumption. The binge eating often continues until the individual is uncomfortably, or even painfully, full. Binge eating is typically triggered by dysphoric mood states, interpersonal stressors, intense hunger following dietary restraint, or feelings related to body weight, body shape, and food. Binge eating may transiently reduce dysphoria, but disparaging self-criticism and depressed mood often follow.

An episode of binge eating is also accompanied by a sense of lack of control (Criterion A2). An individual may be in a frenzied state while binge eating, especially early in the course of the disorder. Some individuals describe a dissociative quality during, or following, the binge episodes. After Bulimia Nervosa has persisted for some time, individuals may report that their binge-eating episodes are no longer characterized by an acute feeling of loss of control, but rather by behavioral indicators of impaired control, such as difficulty resisting binge eating or difficulty stopping a binge once it has begun. The impairment in control associated with binge eating in Bulimia Nervosa is not absolute; for example, an individual may continue binge eating while the telephone is ringing, but will cease if a roommate or spouse unexpectedly enters the room.

Another essential feature of Bulimia Nervosa is the recurrent use of inappropriate compensatory behaviors to prevent weight gain (Criterion B). Many individuals with Bulimia Nervosa employ several methods in their attempt to compensate for binge eating. The most common compensatory technique is the induction of vomiting after an episode of binge eating. This method of purging is employed by 80%–90% of individuals with Bulimia Nervosa who present for treatment at eating disorders clinics. The immediate effects of vomiting include relief from physical discomfort and reduction of fear of gaining weight. In some cases, vomiting becomes a goal in itself, and the person will binge in order to vomit or will vomit after eating a small amount of food. Individuals with Bulimia Nervosa may use a variety of methods to induce vomiting, including the use of fingers or instruments to stimulate the gag reflex. Individuals generally become adept at inducing vomiting and are eventually able to vomit at will. Rarely, individuals consume syrup of ipecac to induce vomiting. Other purging behaviors include the misuse of laxatives and diuretics. Approximately one-third of those with Bulimia Nervosa misuse laxatives after binge eating. Rarely, individuals with the disorder will misuse enemas following episodes of binge eating, but this is seldom the sole compensatory method employed.

Individuals with Bulimia Nervosa may fast for a day or more or exercise excessively in an attempt to compensate for binge eating. Exercise may be considered to be excessive when it significantly interferes with important activities, when it occurs at inappropriate times or in inappropriate settings, or when the individual continues to

exercise despite injury or other medical complications. Rarely, individuals with this disorder may take thyroid hormone in an attempt to avoid weight gain. Individuals with diabetes mellitus and Bulimia Nervosa may omit or reduce insulin doses in order to reduce the metabolism of food consumed during eating binges.

Individuals with Bulimia Nervosa place an excessive emphasis on body shape and weight in their self-evaluation, and these factors are typically the most important ones in determining self-esteem (Criterion D). Individuals with this disorder may closely resemble those with Anorexia Nervosa in their fear of gaining weight, in their desire to lose weight, and in the level of dissatisfaction with their bodies. However, a diagnosis of Bulimia Nervosa should not be given when the disturbance occurs only during episodes of Anorexia Nervosa (Criterion E).

Subtypes

The following subtypes can be used to specify the presence or absence of regular use of purging methods as a means to compensate for the binge eating:

Purging Type. This subtype describes presentations in which the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas during the current episode.

Nonpurging Type. This subtype describes presentations in which the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas during the current episode.

Associated Features and Disorders

Associated descriptive features and mental disorders. Individuals with Bulimia Nervosa typically are within the normal weight range, although some may be slightly underweight or overweight. The disorder occurs but is uncommon among moderately and morbidly obese individuals. There are suggestions that, prior to the onset of the Eating Disorder, individuals with Bulimia Nervosa are more likely to be overweight than their peers. Between binges, individuals with Bulimia Nervosa typically restrict their total caloric consumption and preferentially select low-calorie ("diet") foods while avoiding foods they perceive to be fattening or likely to trigger a binge.

There is an increased frequency of depressive symptoms (e.g., low self-esteem) or Mood Disorders (particularly Dysthymic Disorder and Major Depressive Disorder) in individuals with Bulimia Nervosa. In many or most individuals, the mood disturbance begins at the same time as or following the development of Bulimia Nervosa, and individuals often ascribe their mood disturbances to Bulimia Nervosa. However, in some individuals, the mood disturbance clearly precedes the development of Bulimia Nervosa. There may also be an increased frequency of anxiety symptoms (e.g., fear of social situations) or Anxiety Disorders. These mood and anxiety disturbances frequently remit following effective treatment of Bulimia Nervosa. The lifetime prevalence of Substance Abuse or Dependence, particularly involving alcohol or stimulants, is at least 30% among individuals with Bulimia Nervosa. Stimulant use often begins in an attempt to control appetite and weight. A substantial portion of individ-

uals with Bulimia Nervosa also have personality features that meet criteria for one or more Personality Disorders (most frequently Borderline Personality Disorder).

Preliminary evidence suggests that individuals with Bulimia Nervosa, Purging Type, show more symptoms of depression and greater concern with shape and weight than individuals with Bulimia Nervosa, Nonpurging Type.

Associated laboratory findings. Frequent purging behavior of any kind can produce fluid and electrolyte abnormalities, most frequently hypokalemia, hyponatremia, and hypochloremia. The loss of stomach acid through vomiting may produce a metabolic alkalosis (elevated serum bicarbonate), and the frequent induction of diarrhea through laxative abuse can cause metabolic acidosis. Some individuals with Bulimia Nervosa exhibit mildly elevated levels of serum amylase, probably reflecting an increase in the salivary isoenzyme.

Associated physical examination findings and general medical conditions. Recurrent vomiting eventually leads to a significant and permanent loss of dental enamel, especially from lingual surfaces of the front teeth. These teeth may become chipped and appear ragged and "moth-eaten." There may also be an increased frequency of dental cavities. In some individuals, the salivary glands, particularly the parotid glands, may become notably enlarged. Individuals who induce vomiting by manually stimulating the gag reflex may develop calluses or scars on the dorsal surface of the hand from repeated trauma from the teeth. Serious cardiac and skeletal myopathies have been reported among individuals who regularly use syrup of ipecac to induce vomiting.

Menstrual irregularity or amenorrhea sometimes occurs among females with Bulimia Nervosa; whether such disturbances are related to weight fluctuations, to nutritional deficiencies, or to emotional stress is uncertain. Individuals who chronically abuse laxatives may become dependent on their use to stimulate bowel movements. The fluid and electrolyte disturbances resulting from the purging behavior are sometimes sufficiently severe to constitute medically serious problems. Rare but potentially fatal complications include esophageal tears, gastric rupture, and cardiac arrhythmias. Rectal prolapse has also been reported among individuals with this disorder. Compared with individuals with Bulimia Nervosa, Nonpurging Type, those with the Purging Type are much more likely to have physical problems such as fluid and electrolyte disturbances.

Specific Culture, Age, and Gender Features

Bulimia Nervosa has been reported to occur with roughly similar frequencies in most industrialized countries, including the United States, Canada, Europe, Australia, Japan, New Zealand, and South Africa. Few studies have examined the prevalence of Bulimia Nervosa in other cultures. In clinical studies of Bulimia Nervosa in the United States, individuals presenting with this disorder are primarily white, but the disorder has also been reported among other ethnic groups.

In clinic and population samples, at least 90% of individuals with Bulimia Nervosa are female. Some data suggest that males with Bulimia Nervosa have a higher prevalence of premorbid obesity than do females with Bulimia Nervosa.

Prevalence

The lifetime prevalence of Bulimia Nervosa among women is approximately 1%–3%; the rate of occurrence of this disorder in males is approximately one-tenth of that in females.

Course

Bulimia Nervosa usually begins in late adolescence or early adult life. The binge eating frequently begins during or after an episode of dieting. Disturbed eating behavior persists for at least several years in a high percentage of clinic samples. The course may be chronic or intermittent, with periods of remission alternating with recurrences of binge eating. However, over longer-term follow-up, the symptoms of many individuals appear to diminish. Periods of remission longer than 1 year are associated with better long-term outcome.

Familial Pattern

Several studies have suggested an increased frequency of Bulimia Nervosa, of Mood Disorders, and of Substance Abuse and Dependence in the first-degree biological relatives of individuals with Bulimia Nervosa. A familial tendency toward obesity may exist, but this has not been definitively established.

Differential Diagnosis

Individuals whose binge-eating behavior occurs only during Anorexia Nervosa are given the diagnosis **Anorexia Nervosa, Binge-Eating/Purging Type,** and should *not* be given the additional diagnosis of Bulimia Nervosa. For an individual who binges and purges but whose presentation no longer meets the full criteria for Anorexia Nervosa, Binge-Eating/Purging Type (e.g., when weight is normal or menses have become regular), it is a matter of clinical judgment whether the most appropriate current diagnosis is Anorexia Nervosa, Binge-Eating/Purging Type, In Partial Remission, or Bulimia Nervosa.

In certain neurological or other general medical conditions, such as **Kleine-Levin syndrome,** there is disturbed eating behavior, but the characteristic psychological features of Bulimia Nervosa, such as overconcern with body shape and weight, are not present. Overeating is common in **Major Depressive Disorder, With Atypical Features,** but such individuals do not engage in inappropriate compensatory behavior and do not exhibit the characteristic overconcern with body shape and weight. If criteria for both disorders are met, both diagnoses should be given. Binge-eating behavior is included in the impulsive behavior criterion that is part of the definition of **Borderline Personality Disorder.** If the full criteria for both disorders are met, both diagnoses can be given.

Diagnostic criteria for 307.51 Bulimia Nervosa

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

   (1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances

   (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.

Specify type:

Purging Type: during the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas

Nonpurging Type: during the current episode of Bulimia Nervosa, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas

307.50 Eating Disorder Not Otherwise Specified

The Eating Disorder Not Otherwise Specified category is for disorders of eating that do not meet the criteria for any specific Eating Disorder. Examples include

1. For females, all of the criteria for Anorexia Nervosa are met except that the individual has regular menses.
2. All of the criteria for Anorexia Nervosa are met except that, despite significant weight loss, the individual's current weight is in the normal range.
3. All of the criteria for Bulimia Nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months.
4. The regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food (e.g., self-induced vomiting after the consumption of two cookies).
5. Repeatedly chewing and spitting out, but not swallowing, large amounts of food.

6. Binge-eating disorder: recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of Bulimia Nervosa (see p. 785 for suggested research criteria).

Sleep Disorders

The sleep disorders are organized into four major sections according to presumed etiology. **Primary Sleep Disorders** are those in which none of the etiologies listed below (i.e., another mental disorder, a general medical condition, or a substance) is responsible. Primary Sleep Disorders are presumed to arise from endogenous abnormalities in sleep-wake generating or timing mechanisms, often complicated by conditioning factors. Primary Sleep Disorders in turn are subdivided into **Dyssomnias** (characterized by abnormalities in the amount, quality, or timing of sleep) and **Parasomnias** (characterized by abnormal behavioral or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions).

Sleep Disorder Related to Another Mental Disorder involves a prominent complaint of sleep disturbance that results from a diagnosable mental disorder (often a Mood Disorder or Anxiety Disorder) but that is sufficiently severe to warrant independent clinical attention. Presumably, the pathophysiological mechanisms responsible for the mental disorder also affect sleep-wake regulation.

Sleep Disorder Due to a General Medical Condition involves a prominent complaint of sleep disturbance that results from the direct physiological effects of a general medical condition on the sleep-wake system.

Substance-Induced Sleep Disorder involves prominent complaints of sleep disturbance that result from the concurrent use, or recent discontinuation of use, of a substance (including medications).

The systematic assessment in individuals who present with prominent complaints of sleep disturbance includes an evaluation of the specific type of sleep complaint and a consideration of concurrent mental disorders, general medical conditions, and substance (including medication) use that may be responsible for the sleep disturbance.

Five distinct sleep stages can be measured by polysomnography: rapid eye movement (REM) sleep and four stages of non–rapid eye movement (NREM) sleep (stages 1, 2, 3, and 4). Stage 1 NREM sleep is a transition from wakefulness to sleep and occupies about 5% of time spent asleep in healthy adults. Stage 2 NREM sleep, which is characterized by specific EEG waveforms (sleep spindles and K complexes), occupies about 50% of time spent asleep. Stages 3 and 4 NREM sleep (also known collectively as slow-wave sleep) are the deepest levels of sleep and occupy about 10%–20% of sleep time. REM sleep, during which the majority of typical storylike dreams occur, occupies about 20%–25% of total sleep.

These sleep stages have a characteristic temporal organization across the night. NREM stages 3 and 4 tend to occur in the first one-third to one-half of the night and increase in duration in response to sleep deprivation. REM sleep occurs cyclically throughout the night, alternating with NREM sleep about every 80–100 minutes. REM sleep periods increase in duration toward the morning. Human sleep also varies characteristically across the life span. After relative stability with large amounts of slow-wave sleep in childhood and early adolescence, sleep continuity and depth de-

teriorate across the adult age range. This deterioration is reflected by increased wakefulness and stage 1 sleep and decreased stages 3 and 4 sleep. Because of this, age must be considered in the diagnosis of a Sleep Disorder in any individual.

Polysomnography is the monitoring of multiple electrophysiological parameters during sleep and generally includes measurement of EEG activity, electrooculographic activity, and electromyographic activity. Additional polysomnographic measures may include oral or nasal airflow, respiratory effort, chest and abdominal wall movement, oxyhemoglobin saturation, or exhaled carbon dioxide concentration; these measures are used to monitor respiration during sleep and to detect the presence and severity of sleep apnea. Measurement of peripheral electromyographic activity may be used to detect abnormal movements during sleep. Most polysomnographic studies are conducted during the person's usual sleeping hours—that is, at night. However, daytime polysomnographic studies also are used to quantify daytime sleepiness. The most common daytime procedure is the Multiple Sleep Latency Test (MSLT), in which the individual is instructed to lie down in a dark room and not resist falling asleep; this protocol is repeated five times during the day. Sleep latency (the amount of time required to fall asleep) is measured on each trial and is used as an index of physiological sleepiness. The converse of the MSLT is also used: In the Maintenance of Wakefulness Test (MWT), the individual is placed in a quiet, dimly lit room and instructed to remain awake; this protocol is repeated several times during the day. Again, sleep latency is measured, but it is used here as an index of the individual's ability to maintain wakefulness.

Standard terminology for polysomnographic measures is used throughout the text in this section. *Sleep continuity* refers to the overall balance of sleep and wakefulness during a night of sleep. "Better" sleep continuity indicates consolidated sleep with little wakefulness; "worse" sleep continuity indicates disrupted sleep with more wakefulness. Specific sleep continuity measures include *sleep latency*—the amount of time required to fall asleep (expressed in minutes); *intermittent wakefulness*—the amount of awake time after initial sleep onset (expressed in minutes); and *sleep efficiency*—the ratio of actual time spent asleep to time spent in bed (expressed as a percentage, with higher numbers indicating better sleep continuity). *Sleep architecture* refers to the amount and distribution of specific sleep stages. Sleep architecture measures include absolute amounts of REM sleep and each NREM sleep stage (in minutes), relative amount of REM sleep and NREM sleep stages (expressed as a percentage of total sleep time), and latency between sleep onset and the first REM period (REM latency).

The text for each of the Sleep Disorders contains a section describing its relationship to corresponding disorders in the *The International Classification of Sleep Disorders: (ICSD) Diagnostic and Coding Manual*, published in 1990 by the American Sleep Disorders Association.

Primary Sleep Disorders

Dyssomnias

Dyssomnias are primary disorders of initiating or maintaining sleep or of excessive sleepiness and are characterized by a disturbance in the amount, quality, or timing of

sleep. This section includes Primary Insomnia, Primary Hypersomnia, Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, and Dyssomnia Not Otherwise Specified.

307.42 Primary Insomnia

Diagnostic Features

The essential feature of Primary Insomnia is a complaint of difficulty initiating or maintaining sleep or of nonrestorative sleep that lasts for at least 1 month (Criterion A) and causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion B). The disturbance in sleep does not occur exclusively during the course of another sleep disorder (Criterion C) or mental disorder (Criterion D) and is not due to the direct physiological effects of a substance or a general medical condition (Criterion E).

Individuals with Primary Insomnia most often report a combination of difficulty falling asleep and intermittent wakefulness during sleep. The specific type of sleep complaint often varies over time. For instance, individuals who complain of difficulty falling asleep at one time may later complain of difficulty maintaining sleep, and vice versa. Less commonly, individuals with Primary Insomnia may complain only of nonrestorative sleep—that is, feeling that their sleep was restless, light, or of poor quality. Not all individuals with nighttime sleep disturbances are distressed or have functional impairment. A diagnosis of Primary Insomnia should be reserved for those individuals with significant distress or impairment.

Primary Insomnia is often associated with increased physiological, cognitive, or emotional arousal in combination with negative conditioning for sleep. A marked preoccupation with and distress due to the inability to sleep may contribute to the development of a vicious cycle: the more the individual strives to sleep, the more frustrated and distressed he or she becomes and the less he or she is able to sleep. Lying in a bed in which the individual has frequently spent sleepless nights may cause frustration and conditioned arousal. Conversely, the individual may fall asleep more easily when not trying to do so (e.g., while watching television, reading, or riding in a car). Some individuals with increased arousal and negative conditioning report that they sleep better away from their own bedrooms and their usual routines. Individuals with Primary Insomnia may thereby acquire maladaptive sleep habits (e.g., daytime napping, spending excessive time in bed, following an erratic sleep schedule, performing sleep-incompatible behaviors in bed) during the course of the disorder. Chronic insomnia may lead to decreased feelings of well-being during the day (e.g., deterioration of mood and motivation; decreased attention, energy, and concentration; and an increase in fatigue and malaise). Although individuals often have the subjective complaint of daytime fatigue, polysomnographic studies usually do not demonstrate an increase in physiological signs of sleepiness.

Associated Features and Disorders

Associated descriptive features and mental disorders. Many individuals with Primary Insomnia have a history of "light" or easily disturbed sleep prior to the devel-

opment of more persistent sleep problems. Other associated factors may include anxious overconcern with general health and increased sensitivity to the daytime effects of mild sleep loss. Symptoms of anxiety or depression that do not meet criteria for a specific mental disorder may be present. Interpersonal, social, and occupational problems may develop as a result of overconcern with sleep, increased daytime irritability, and poor concentration. Problems with inattention and concentration may also lead to accidents. Individuals with severe insomnia have greater functional impairment, lower productivity, and increased health care utilization compared with individuals without sleep complaints. Individuals with Primary Insomnia may also report interpersonal and work-related stress.

Individuals with Primary Insomnia may have a history of mental disorders, particularly Mood Disorders and Anxiety Disorders. Primary Insomnia also constitutes a risk factor for (or perhaps an early symptom of) subsequent Mood Disorders, Anxiety Disorders, and Substance Use Disorders. Individuals with Primary Insomnia sometimes use medications inappropriately: hypnotics or alcohol to help with nighttime sleep, anxiolytics to combat tension or anxiety, and caffeine or other stimulants to combat excessive fatigue. In some cases, this type of substance use may progress to Substance Abuse or Substance Dependence.

Associated laboratory findings. Polysomnography often demonstrates poor sleep continuity (e.g., increased sleep latency, increased intermittent wakefulness, and decreased sleep efficiency) and may demonstrate increased stage 1 sleep and decreased stages 3 and 4 sleep. Other laboratory findings may include increased muscle tension and increased amounts of alpha and beta activity during sleep as measured by quantitative EEG analysis. These features must be interpreted within the context of age-appropriate norms. Polysomnographic measures often show considerable variability from night to night. Individuals with Primary Insomnia may also have substantial discrepancies between subjective and polysomnographic measures of sleep quantity, most commonly in the direction of underestimating sleep amount. Some individuals may report better sleep in the laboratory than at home, suggesting a conditioned basis for sleep complaints. Individuals with Primary Insomnia typically do not have increased daytime sleepiness as measured by sleep laboratory testing compared with individuals without any Sleep Disorders. Other psychophysiological tests may also show high arousal (e.g., increased muscle tension, excessive physiological reactivity to stress, and increased metabolic rate).

Individuals with Primary Insomnia may also have elevated scores on self-report psychological or personality inventories (e.g., on profiles indicating chronic, mild depression and anxiety; an "internalizing" style of conflict resolution; and a somatic focus). Measures of neuropsychological test performance do not show consistent patterns of impairment among individuals with Primary Insomnia.

Associated physical examination findings and general medical conditions. Individuals with Primary Insomnia may appear fatigued or haggard but show no other characteristic abnormalities on physical examination. There may be an increased incidence of stress-related psychophysiological problems (e.g., tension headache, increased muscle tension, gastric distress).

Specific Age and Gender Features

Survey data consistently demonstrate that complaints of insomnia are more prevalent with increasing age and among women. The increasing prevalence of insomnia complaints with age may be attributable partly to the increased rates of physical health problems in the elderly. Young adults more often complain of difficulty falling asleep, whereas midlife and elderly adults are more likely to have difficulty with maintaining sleep and early morning awakening. Paradoxically, despite the greater prevalence of insomnia complaints among elderly women, polysomnographic studies generally indicate better preservation of sleep continuity and slow-wave sleep in elderly females than in elderly males. The reason for this discrepancy between self-report and laboratory data is not known. Although polysomnographic studies are of limited value in the routine evaluation of insomnia, they may be more useful in the differential diagnosis of insomnia among older adults than among younger individuals. This is because older individuals more often have identifiable etiologies for their sleep complaints, such as periodic limb movements and sleep apnea.

Prevalence

There are few data regarding the prevalence of Primary Insomnia in the general population. Population surveys indicate a 1-year prevalence of insomnia complaints of 30%–45% in adults. The prevalence of Primary Insomnia is approximately 1%–10% in the general adult population and up to 25% in the elderly. In clinics specializing in sleep disorders, approximately 15%–25% of individuals with chronic insomnia are diagnosed with Primary Insomnia.

Course

The factors that precipitate Primary Insomnia may differ from those that perpetuate it. Most cases have a fairly sudden onset at a time of psychological, social, or medical stress. Primary Insomnia often persists long after the original causative factors resolve, due to the development of heightened arousal and negative conditioning. For example, a person with a painful injury who spends a great deal of time in bed and has difficulty sleeping may then develop negative associations for sleep. Negative associations, increased arousal, and conditioned awakenings may then persist beyond the convalescent period, leading to Primary Insomnia. A similar scenario may develop in association with insomnia that occurs in the context of an acute psychological stress or a mental disorder. For instance, insomnia that occurs during an episode of Major Depressive Disorder can become a focus of attention with consequent negative conditioning, and insomnia may persist long after resolution of the depressive episode. In some cases, Primary Insomnia may develop gradually without a clear stressor.

Primary Insomnia typically begins in young adulthood or middle age and is rare in childhood or adolescence. In exceptional cases, the insomnia can be documented back to childhood. The course of Primary Insomnia is variable. It may be limited to a period of several months, particularly if precipitated by a psychosocial or general medical stressor that later resolves. However, approximately 50%–75% of individuals

with insomnia complaints have chronic symptoms lasting for more than 1 year, and previous insomnia is the strongest single risk factor for current insomnia. Some individuals experience an episodic course, with periods of better or worse sleep occurring in response to life events such as vacations or stress.

Familial Pattern

The predisposition toward light and disrupted sleep has a familial association. Limited data from twin studies have yielded inconsistent results regarding the importance of genetic factors in Primary Insomnia.

Differential Diagnosis

"Normal" sleep duration varies considerably in the general population. Some individuals who require little sleep ("short sleepers") may be concerned about their sleep duration. **Short sleepers** are distinguished from those with Primary Insomnia by their lack of difficulty falling asleep and by the absence of characteristic symptoms of Primary Insomnia (e.g., intermittent wakefulness, fatigue, concentration problems, or irritability). However, some short sleepers are uninformed as to their abbreviated biological need for sleep, and in their attempt to prolong time in bed, they create an insomnia sleep pattern.

Daytime sleepiness, which is a characteristic feature of **Primary Hypersomnia,** may infrequently occur in Primary Insomnia but is not as severe. When daytime sleepiness is judged to be due to insomnia, an additional diagnosis of Primary Hypersomnia is not given.

Jet Lag and Shift Work Types of **Circadian Rhythm Sleep Disorder** are distinguished from Primary Insomnia by the history of recent transmeridian travel or shift work. Individuals with the Delayed Sleep Phase Type of Circadian Rhythm Sleep Disorder report sleep-onset insomnia only when they try to sleep at socially normal times, but they do not report difficulty falling asleep or staying asleep when they sleep at their preferred times.

Narcolepsy may cause insomnia complaints, particularly in older adults. However, Narcolepsy rarely involves a major complaint of insomnia and is distinguished from Primary Insomnia by symptoms of prominent daytime sleepiness, cataplexy, sleep paralysis, and sleep-related hallucinations.

Breathing-Related Sleep Disorder, particularly central sleep apnea, may involve a complaint of chronic insomnia and daytime impairment. However, clinically significant sleep apnea is an uncommon finding among otherwise healthy young and middle-aged individuals with chronic insomnia (although it may be more common in the elderly). A careful history may reveal periodic pauses in breathing during sleep or crescendo-decrescendo breathing (Cheyne-Stokes respiration). A history of central nervous system injury or disease may further suggest a Breathing-Related Sleep Disorder. Polysomnography can confirm the presence of apneic events. Most individuals with Breathing-Related Sleep Disorder have obstructive apnea that can be distinguished from Primary Insomnia by a history of loud snoring, breathing pauses during sleep, and excessive daytime sleepiness.

Parasomnias are characterized by a complaint of unusual behavior or events dur-

ing sleep that sometimes may lead to intermittent awakenings. However, it is these behavioral events that dominate the clinical picture in a Parasomnia rather than the insomnia.

Primary Insomnia must be distinguished from **mental disorders that include insomnia as an essential or associated feature** (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, Schizophrenia). The diagnosis of Primary Insomnia is not given if insomnia occurs exclusively during the course of another mental disorder. A thorough investigation for the presence of other mental disorders is essential before considering the diagnosis of Primary Insomnia. A diagnosis of Primary Insomnia can be made in the presence of another current or past mental disorder if the mental disorder is judged to not account for the insomnia or if the insomnia and the mental disorder have an independent course. When insomnia occurs as a manifestation of, and exclusively during the course of, another mental disorder (e.g., a Mood, Anxiety, Somatoform, or Psychotic Disorder), the diagnosis of **Insomnia Related to Another Mental Disorder** may be more appropriate. This diagnosis should only be considered when the insomnia is the predominant complaint and is sufficiently severe to warrant independent clinical attention; otherwise, no separate diagnosis is necessary. Clinical features such as negative conditioning and poor sleep hygiene are more consistent with a diagnosis of Primary Insomnia, whereas clinically significant nonsleep symptoms (e.g., depressed mood, anxiety) and a chronic, severe course of insomnia are more common in individuals with Insomnia Related to Another Mental Disorder.

In clinical settings, polysomnography is not typically useful in the differential diagnosis of Primary Insomnia versus Insomnia Related to Another Mental Disorder.

Primary Insomnia must be distinguished from **Sleep Disorder Due to a General Medical Condition, Insomnia Type.** The diagnosis should be Sleep Disorder Due to a General Medical Condition when the insomnia is judged to be the direct physiological consequence of a specific general medical condition (e.g., pheochromocytoma, hyperthyroidism, congestive heart failure, chronic obstructive pulmonary disease) (see p. 651). This determination is based on history, laboratory findings, or physical examination. **Substance-Induced Sleep Disorder, Insomnia Type,** is distinguished from Primary Insomnia by the fact that a substance (i.e., a drug of abuse, a medication, or exposure to a toxin) is judged to be etiologically related to the insomnia (see p. 655). For example, insomnia occurring only in the context of heavy coffee consumption would be diagnosed as Caffeine-Induced Sleep Disorder, Insomnia Type, With Onset During Intoxication.

Relationship to International Classification of Sleep Disorders

Primary Insomnia subsumes a number of insomnia diagnoses in the International Classification of Sleep Disorders (ICSD), including Psychophysiological Insomnia, Sleep State Misperception, Idiopathic Insomnia, and some cases of Inadequate Sleep Hygiene. Psychophysiological Insomnia most closely resembles Primary Insomnia, particularly in terms of arousal and conditioning factors. Sleep State Misperception is a condition characterized by complaints of insomnia with a marked discrepancy between subjective and objective estimates of sleep. Idiopathic Insomnia includes those cases with onset in childhood and a lifelong course, presumably due to an abnormality in the neurological control of the sleep-wake system. Inadequate Sleep Hygiene

refers to insomnia resulting from behavioral practices that increase arousal or disrupt sleep organization (e.g., working late into the night, taking excessive daytime naps, or keeping irregular sleep hours).

Diagnostic criteria for 307.42 Primary Insomnia

A. The predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month.

B. The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The sleep disturbance does not occur exclusively during the course of Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia.

D. The disturbance does not occur exclusively during the course of another mental disorder (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, a delirium).

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

307.44 Primary Hypersomnia

Diagnostic Features

The essential feature of Primary Hypersomnia is excessive sleepiness for at least 1 month as evidenced either by prolonged sleep episodes or by daytime sleep episodes occurring almost daily (Criterion A). The excessive sleepiness must be sufficiently severe to cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion B). The excessive sleepiness does not occur exclusively during the course of another Sleep Disorder (Criterion C) or mental disorder (Criterion D) and is not due to the direct physiological effects of a substance or a general medical condition (Criterion E).

In individuals with Primary Hypersomnia, the duration of the major sleep episode (for most individuals, nocturnal sleep) may range from 8 to 12 hours and is often followed by difficulty awakening in the morning. The actual quality of nocturnal sleep is normal. Excessive sleepiness during normal waking hours takes the form of intentional naps or inadvertent episodes of sleep. Objective measurements demonstrate increased physiological sleepiness. Daytime naps tend to be relatively long (often lasting an hour or more), are experienced as unrefreshing, and often do not lead to improved alertness. Individuals typically feel sleepiness developing over a period of time, rather than experiencing a sudden sleep "attack." Unintentional sleep episodes typically occur in low-stimulation and low-activity situations (e.g., while attending lectures, reading, watching television, or driving long distances).

Hypersomnia can lead to significant distress and dysfunction in work and social relationships. Prolonged nocturnal sleep and difficulty awakening can result in diffi-

culty in meeting morning obligations. Unintentional daytime sleep episodes can be embarrassing and even dangerous, if, for instance, the individual is driving or operating machinery when the episode occurs. The low level of alertness that occurs while an individual fights sleepiness can lead to poor efficiency, poor concentration, and poor memory during daytime activities. Sleepiness, often misattributed to boredom or laziness, can also disrupt social and family relationships.

Specifier

Recurrent. This specifier is used if there are periods of excessive sleepiness that last at least 3 days occurring several times a year for at least 2 years.

Most individuals with Primary Hypersomnia have consistent and persistent symptoms. In contrast, the Recurrent form should be noted if symptoms occur periodically for several days to several weeks, with symptomatic periods recurring several times per year. Between periods of excessive sleepiness, sleep duration and daytime alertness are normal. In the recurrent form of Primary Hypersomnia known as Kleine-Levin syndrome, individuals may spend 18–20 hours asleep or in bed. The recurrent periods of sleepiness are associated with other characteristic clinical features indicating disinhibition. Indiscriminate hypersexuality including inappropriate sexual advances and overt masturbation can be seen in males (and less often in females). Compulsive overeating with acute weight gain may occur. Irritability, depersonalization, depression, confusion, and occasional hallucinations have been described in some individuals, and impulsive behaviors can also occur. Other recurrent forms of hypersomnia can be seen in the absence of these features. For instance, some females report regularly occurring periods of hypersomnia at specific times of their menstrual cycle.

Associated Features and Disorders

Associated descriptive features and mental disorders. In Primary Hypersomnia, sleep tends to be continuous but nonrestorative. Individuals with this disorder fall asleep quickly and have good sleep efficiency, but may have difficulty waking up in the morning, sometimes appearing confused, combative, or ataxic. This prolonged impairment of alertness at the sleep-wake transition is often referred to as "sleep drunkenness."

Persistent daytime sleepiness can lead to automatic behavior (usually of a very routine, low-complexity type) that the individual carries out with little or no subsequent recall. For example, individuals may find themselves having driven several miles from where they thought they were, unaware of the "automatic" driving they did in the preceding minutes.

Although precise data are not available regarding comorbidity with mental disorders, many individuals with Primary Hypersomnia have symptoms of depression that may meet criteria for a Mood Disorder. This may be related to the psychosocial consequences of excessive sleepiness. Individuals with hypersomnia are also at risk for Substance-Related Disorders, particularly related to self-medication with stimulants.

Associated laboratory findings. In Primary Hypersomnia, nocturnal polysomnography demonstrates a normal to prolonged sleep duration, short sleep latency, normal to increased sleep continuity, and normal distributions of rapid eye movement (REM) and non–rapid eye movement (NREM) sleep. Some individuals with this disorder may have increased amounts of slow-wave sleep. Increased spindle density during stage 2 sleep may be present. Sleep-onset REM periods (the occurrence of REM sleep within 20 minutes of sleep onset), breathing-related sleep disturbances, and frequent limb movements disrupting sleep are not present. The Multiple Sleep Latency Test (MSLT) documents excessive physiological daytime sleepiness, typically indicated by mean sleep latency values of 5–10 minutes. REM sleep does not occur during the daytime sleep episodes. Nocturnal polysomnography and the MSLT do not reveal findings characteristic of other causes of hypersomnia.

In the Recurrent Kleine-Levin form of Primary Hypersomnia, routine EEG studies performed during the periods of hypersomnia show general slowing of the background rhythm and paroxysmal bursts of theta activity. Nocturnal polysomnography shows an increase in total sleep time and short REM sleep latency. MSLT studies confirm increased physiological sleepiness, with sleep latencies generally less than 10 minutes. Sleep-onset REM periods may be seen during symptomatic periods.

Associated physical examination findings and general medical conditions. Individuals with Primary Hypersomnia often appear sleepy and may even fall asleep in the clinician's waiting area. A subset of individuals with Primary Hypersomnia have a family history of hypersomnia and also have symptoms of autonomic nervous system dysfunction, including recurrent vascular-type headaches, reactivity of the peripheral vascular system (Raynaud's phenomenon), and fainting. Individuals with the Recurrent Kleine-Levin form may have nonspecific neurological examination findings including depressed deep tendon reflexes, dysarthria, and nystagmus.

Specific Age or Gender Features

Hyperactivity may be one of the presenting signs of daytime sleepiness in children. Voluntary napping increases with age, but this normal phenomenon is distinct from Primary Hypersomnia. Kleine-Levin syndrome affects males about three times more often than it affects females.

Prevalence

The true prevalence of Primary Hypersomnia in the general population is not known. Approximately 5%–10% of individuals who present to sleep disorders clinics with complaints of daytime sleepiness are diagnosed as having Primary Hypersomnia. The Recurrent form of Primary Hypersomnia known as Kleine-Levin syndrome is rare. Population surveys find a complaint of daytime sleepiness in 0.5%–5.0% of adults, without regard to specific causes or diagnoses. After other common causes are accounted for, the lifetime prevalence of clinically significant hypersomnia is at least 16%, and the incidence over approximately a 4-year interval is about 8%.

Course

Primary Hypersomnia typically begins between ages 15 and 30 years, with a gradual progression over weeks to months. For most individuals, the course is then chronic and stable, unless treatment is initiated. The development of other sleep disorders (e.g., Breathing-Related Sleep Disorder) may worsen the degree of sleepiness. Kleine-Levin syndrome also begins during adolescence and may continue its periodic course for decades, although it often resolves during middle age.

Familial Pattern

The subgroup of individuals with autonomic dysfunction are more likely than other individuals with Primary Hypersomnia to have family members with Primary Hypersomnia. Kleine-Levin syndrome does not demonstrate familial aggregation.

Differential Diagnosis

"Normal" sleep duration varies considerably in the general population. **"Long sleepers"** (i.e., individuals who require a greater than average amount of sleep) do not have excessive daytime sleepiness, sleep drunkenness, or automatic behavior when they obtain their required amount of nocturnal sleep. If social or occupational demands lead to shorter nocturnal sleep, daytime symptoms may appear. In Primary Hypersomnia, by contrast, symptoms of excessive sleepiness occur regardless of nocturnal sleep duration.

An **inadequate amount of nocturnal sleep** can produce symptoms of daytime sleepiness very similar to those of Primary Hypersomnia. An average sleep duration of fewer than 7 hours per night strongly suggests inadequate nocturnal sleep, and an average of more than 9 hours of sleep per 24-hour period suggests Primary Hypersomnia. Individuals with inadequate nocturnal sleep typically "catch up" with longer sleep durations on days when they are free from social or occupational demands or on vacations. Unlike Primary Hypersomnia, insufficient nocturnal sleep is unlikely to persist unabated for decades. A diagnosis of Primary Hypersomnia should not be made if there is a question regarding the adequacy of nocturnal sleep duration. A diagnostic and therapeutic trial of sleep extension for 10–14 days can often clarify the diagnosis.

Daytime sleepiness, which is a characteristic feature of Primary Hypersomnia, can also occur in **Primary Insomnia,** but the sleepiness or fatigue is less severe in individuals with Primary Insomnia. When daytime sleepiness is judged to be due to insomnia, an additional diagnosis of Primary Hypersomnia is not given.

Primary Hypersomnia and **Narcolepsy** are similar with respect to the degree of daytime sleepiness, age at onset, and stable course over time but can be distinguished based on distinctive clinical and laboratory features. Individuals with Primary Hypersomnia typically have longer and less disrupted nocturnal sleep, greater difficulty awakening, more persistent daytime sleepiness (as opposed to more discrete "sleep attacks" in Narcolepsy), longer and less refreshing daytime sleep episodes, and little or no dreaming during daytime naps. By contrast, individuals with Narcolepsy have cataplexy and recurrent intrusions of elements of REM sleep into the transition be-

tween sleep and wakefulness (e.g., sleep-related hallucinations and sleep paralysis). The MSLT typically demonstrates shorter sleep latencies (i.e., greater physiological sleepiness) as well as the presence of multiple sleep-onset REM periods in individuals with Narcolepsy.

Individuals with Primary Hypersomnia and **Breathing-Related Sleep Disorder** may have similar patterns of excessive sleepiness. Breathing-Related Sleep Disorder is suggested by a history of loud snoring, pauses in breathing during sleep, brain injury, or cardiovascular disease and by the presence of obesity, oropharyngeal anatomical abnormalities, hypertension, or heart failure on physical examination. Polysomnographic studies can confirm the presence of apneic events in Breathing-Related Sleep Disorder (and their absence in Primary Hypersomnia).

Circadian Rhythm Sleep Disorder is often characterized by daytime sleepiness. A history of an abnormal sleep-wake schedule (with shifted or irregular hours) is present in individuals with Circadian Rhythm Sleep Disorder. **Parasomnias** rarely produce the prolonged, undisturbed nocturnal sleep or daytime sleepiness characteristic of Primary Hypersomnia.

Primary Hypersomnia must be distinguished from **mental disorders that include hypersomnia as an essential or associated feature.** In particular, complaints of daytime sleepiness may occur in a **Major Depressive Episode, With Atypical Features,** and in the depressed phase of **Bipolar Disorder.** The diagnosis of Primary Hypersomnia is not given if hypersomnia occurs exclusively during the course of another mental disorder. A thorough investigation for the presence of other mental disorders is essential before considering the diagnosis of Primary Hypersomnia. A diagnosis of Primary Hypersomnia can be made in the presence of another current or past mental disorder if the mental disorder is judged to not account for the hypersomnia or if the hypersomnia and the mental disorder have an independent course (e.g., in an individual with chronic hypersomnia who later develops a Major Depressive Disorder). In contrast, when hypersomnia occurs as a manifestation of, and exclusively during the course of, another mental disorder, the diagnosis of Hypersomnia Related to Another Mental Disorder may be more appropriate. This diagnosis should only be considered when the hypersomnia is the predominant complaint and is sufficiently severe to warrant independent clinical attention; otherwise, no separate diagnosis is necessary. In general, laboratory testing of daytime sleepiness in individuals with Hypersomnia Related to a Mental Disorder often shows normal or only mild levels of physiological sleepiness compared with individuals with Primary Hypersomnia.

Primary Hypersomnia must be distinguished from **Sleep Disorder Due to a General Medical Condition, Hypersomnia Type.** The diagnosis is Sleep Disorder Due to a General Medical Condition when the hypersomnia is judged to be a direct physiological consequence of a specific general medical condition (e.g., morbid obesity, brain tumor) (see p. 651). This determination is based on history, laboratory findings, or physical examination. **Substance-Induced Sleep Disorder, Hypersomnia Type,** is distinguished from Primary Hypersomnia by the fact that a substance (i.e., a drug of abuse, a medication, or exposure to a toxin) is judged to be etiologically related to the hypersomnia (see p. 655). For example, hypersomnia occurring only in the context of withdrawal from cocaine would be diagnosed as Cocaine-Induced Sleep Disorder, Hypersomnia Type, With Onset During Withdrawal.

Relationship to the International Classification of Sleep Disorders

Primary Hypersomnia is analogous to the diagnosis of Idiopathic Hypersomnia in the International Classification of Sleep Disorders (ICSD). In addition, the ICSD includes a separate category for Recurrent Hypersomnia, which is analogous to the Recurrent form of Primary Hypersomnia.

Diagnostic criteria for 307.44 Primary Hypersomnia

A. The predominant complaint is excessive sleepiness for at least 1 month (or less if recurrent) as evidenced by either prolonged sleep episodes or daytime sleep episodes that occur almost daily.

B. The excessive sleepiness causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The excessive sleepiness is not better accounted for by insomnia and does not occur exclusively during the course of another Sleep Disorder (e.g., Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia) and cannot be accounted for by an inadequate amount of sleep.

D. The disturbance does not occur exclusively during the course of another mental disorder.

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Specify if:

Recurrent: if there are periods of excessive sleepiness that last at least 3 days occurring several times a year for at least 2 years

347 Narcolepsy

Diagnostic Features

The essential features of Narcolepsy are repeated irresistible attacks of refreshing sleep, cataplexy, and recurrent intrusions of elements of rapid eye movement (REM) sleep into the transition period between sleep and wakefulness. The individual's sleepiness typically decreases after a sleep attack, only to return several hours later. The sleep attacks must occur daily over a period of at least 3 months to establish the diagnosis (Criterion A), although most individuals describe many years of sleep attacks prior to seeking clinical attention. In addition to sleepiness, individuals with Narcolepsy experience one or both of the following: cataplexy (i.e., episodes of sudden, bilateral, reversible loss of muscle tone that last for seconds to minutes and are usually precipitated by intense emotion) (Criterion B1) or recurrent intrusions of elements of rapid eye movement (REM) sleep into the transition between sleep and wakefulness as manifested by paralysis of voluntary muscles or dreamlike hallucinations (Criterion B2). Many sleep experts allow the diagnosis to be made in the absence

of cataplexy or intrusions of REM sleep elements if the individual demonstrates pathological sleepiness and two or more sleep-onset REM periods during a Multiple Sleep Latency Test (MSLT). The symptoms must not be due to the direct physiological effects of a substance (including a medication) or another general medical condition (Criterion C). Although Narcolepsy is classified in the chapter of ICD devoted to neurological conditions, it is included in this section to assist in differential diagnosis in individuals with excessive sleepiness and is coded on Axis I.

Episodes of sleepiness in Narcolepsy are often described as irresistible, resulting in unintended sleep in inappropriate situations (e.g., while driving an automobile, attending meetings, or carrying on a conversation). Low-stimulation, low-activity situations typically exaggerate the degree of sleepiness (e.g., falling asleep while reading, watching television, or attending lectures). Sleep episodes generally last 10–20 minutes but can last up to an hour if uninterrupted. Dreaming is frequently reported. Individuals have varying abilities to "fight off" these sleep attacks. Some individuals take naps intentionally in order to manage their sleepiness. Individuals with Narcolepsy typically have 2–6 episodes of sleep (intentional and unintentional) per day when untreated. Sleep episodes are usually superimposed on a more normal degree of alertness, although some individuals describe constant sleepiness of some degree.

Cataplexy often develops several years after the onset of daytime sleepiness and occurs in approximately 70% of individuals with the disorder. The loss of muscle tone with cataplexy may be subtle, leading to a sagging jaw or drooping eyelids, head, or arms not noticeable to observers. Cataplexy can also be more dramatic, and the individual may drop objects being carried, buckle at the knees, or actually fall to the ground. Respiratory and eye muscles are not affected. The muscle weakness usually lasts only seconds, although periods of up to a half hour have been reported. Episodes are followed by a full return of normal muscle strength. Full consciousness and alertness are preserved during cataplectic episodes. Individuals can clearly describe events and have no confusion before or after the episode. Rarely, prolonged episodes of cataplexy may lead into sleep episodes. Cataplexy is usually triggered by a strong emotional stimulus (e.g., anger, surprise, laughter). Sleep deprivation typically increases the frequency and severity of episodes of cataplexy.

Approximately 20%–40% of individuals with Narcolepsy also experience intense dreamlike imagery just before falling asleep (hypnagogic hallucinations) or just after awakening (hypnopompic hallucinations). Most sleep-related hallucinations are visual and incorporate elements of the actual environment. For instance, individuals may describe objects appearing through cracks in the wall or describe objects moving in a picture on the wall. The hallucinations may also be auditory (e.g., hearing intruders in the home) or kinetic (e.g., sensation of flying). Although hypnagogic and hypnopompic hallucinations are important symptoms among individuals with Narcolepsy, these symptoms are also present in approximately 10%–15% of the general population. Approximately 30%–50% of individuals with Narcolepsy also experience sleep paralysis just on falling asleep or awakening. In this condition, individuals describe being awake but unable to move or speak. They may also complain of feeling unable to breathe, although the diaphragm is spared and respiration continues. It should be noted, however, that 40%–50% of normal sleepers report having had isolated episodes of sleep paralysis at least once during their lifetime. Sleep-related hallucinations and sleep paralysis may occur simultaneously, resulting in an often

terrifying experience of seeing or hearing unusual things and being unable to move. Both sleep-related hallucinations and sleep paralysis last for seconds to a few minutes and terminate spontaneously. Both phenomena (vivid mental imagery and skeletal muscle atonia) are thought to result from dissociated elements of REM sleep intruding into wakefulness.

Associated Features and Disorders

Associated descriptive features and mental disorders. Some individuals with Narcolepsy experience generalized daytime sleepiness between the discrete sleep attacks. They may describe being able to sleep at any time in any situation. Automatic behavior, in which the individual engages in activity without full awareness, can occur as a result of profound sleepiness. Individuals may drive, converse, or even work during episodes of automatic behavior. Frequent, intense, and vivid dreams may occur during nocturnal sleep. Individuals with Narcolepsy often experience fragmented nighttime sleep as a result of spontaneous awakenings or periodic limb movements. Rarely, individuals may present with a chief complaint of insomnia rather than hypersomnia.

Individuals with Narcolepsy may hesitate to engage in social activities because they fear falling asleep or having an episode of cataplexy. They may also strive to prevent attacks of cataplexy by exerting control over their emotions, which may lead to a generalized lack of expressiveness that interferes with social relations. Narcolepsy can severely limit daytime functioning because of repeated, uncontrollable sleep attacks, automatic behavior, and episodes of cataplexy. Individuals with Narcolepsy are at risk for accidental injury to themselves or others because of falling asleep in dangerous situations (e.g., while driving an automobile or operating machinery).

A concurrent mental disorder or history of another mental disorder can be found in approximately 40% of individuals with Narcolepsy. The most common associated disorders are Mood Disorders (primarily Major Depressive Disorder and Dysthymic Disorder), followed by Substance-Related Disorders and Generalized Anxiety Disorder. A history of Parasomnias such as Sleepwalking Disorder, bruxism (clenching of the jaw and grinding teeth), rapid eye movement (REM) sleep behavior disorder, and Enuresis appears to be more common in individuals with Narcolepsy.

Associated laboratory findings. Findings from the daytime Multiple Sleep Latency Test (MSLT) include an average sleep latency of less than 5 minutes and the appearance of REM sleep during two or more naps on a five-nap MSLT. These MSLT criteria will correctly identify approximately two out of three individuals with Narcolepsy. Nocturnal polysomnographic studies frequently demonstrate sleep latencies of less than 10 minutes and sleep-onset REM periods. Additional findings on polysomnography may include frequent transient arousals, decreased sleep efficiency, increased stage 1 sleep, increased REM sleep, and an increase in the frequency of eye movements within the REM periods ("REM density"). Periodic limb movements and episodes of sleep apnea are often noted, but the latter occur less frequently than in Breathing-Related Sleep Disorder. Individuals with Narcolepsy may demonstrate differences in the amplitude or timing of circadian functioning as measured by core body temperature and motor activity.

Human leukocyte antigen (HLA) typing of individuals with Narcolepsy often shows the presence of HLA-DQB1*0602. This marker is present in almost all individuals with Narcolepsy and cataplexy and is independent of racial ethnicity. However, HLA-DQB1*0602 is present in only 40% of individuals with Narcolepsy without cataplexy and is present in 20%–25% of the general population. Other HLA markers vary in terms of their sensitivity and specificity in different racial groups.

Associated physical examination findings and general medical conditions. Individuals with Narcolepsy may appear sleepy during the clinical interview and examination and may actually fall asleep in the waiting area or examination room. During episodes of cataplexy, individuals may slump in the chair and have slurred speech or drooping eyelids.

Specific Age Features

Hyperactivity may be one of the presenting signs in children with daytime sleepiness. The core clinical features and laboratory findings in Narcolepsy in children are similar to those in adults. However, cataplexy and mild daytime sleepiness may be more difficult to identify in children than in adults.

Prevalence

Epidemiological studies indicate a prevalence of 0.02%–0.16% for Narcolepsy in the adult population, with equal rates in females and males.

Course

Daytime sleepiness is almost always the first symptom of Narcolepsy and usually becomes clinically significant during adolescence. However, on careful review, some degree of sleepiness may have been present even during preschool and early school ages. Onset after age 40 is unusual. However, some individuals with Narcolepsy may not identify excessive sleepiness as a symptom of an illness. This may explain why many individuals are first diagnosed with Narcolepsy many years after the first onset of symptoms. Acute psychosocial stressors or acute alterations in the sleep-wake schedule herald the onset in roughly half of cases. Cataplexy may develop concurrently with sleepiness but often appears months, years, or even decades after the onset of sleepiness. Sleep-related hallucinations and sleep paralysis are more variable symptoms of the disorder and may not occur in some individuals. Disrupted nocturnal sleep usually develops later in the course of the disorder, often when individuals are in their 40s or 50s.

The excessive sleepiness of Narcolepsy has a stable course over time. The development of other Sleep Disorders (e.g., periodic limb movements or Breathing-Related Sleep Disorder) may worsen the degree of sleepiness, whereas treatment with stimulant medications may improve it. Cataplexy usually has a stable course as well, although some individuals report decreased symptoms or even complete cessation of symptoms after many years. Similarly, the sleep-related hallucinations and sleep paralysis may go into remission while the daytime sleepiness and sleep attacks persist.

Familial Pattern

Data from HLA studies and family studies strongly suggest a role for genetic factors in the development of Narcolepsy. The mode of inheritance has not been determined but is likely multifactorial. Approximately 5%–15% of first-degree biological relatives of probands with Narcolepsy have the disorder. Approximately 25%–50% of the first-degree biological relatives of individuals with Narcolepsy have other disorders characterized by excessive sleepiness (such as Primary Hypersomnia).

Differential Diagnosis

Narcolepsy must be differentiated from **normal variations in sleep,** sleep deprivation, other primary Sleep Disorders, and Sleep Disorder Related to Another Mental Disorder, Hypersomnia Type. Many individuals feel some sleepiness during the day, particularly in the afternoon hours when an increase in physiological sleepiness occurs. However, such individuals do not have irresistible sleep at other times of the day and can "fight through" their sleepiness with increased mental and physical effort. They generally do not experience cataplexy, sleep-related hallucinations, or sleep paralysis. Episodes of muscle weakness may occur in individuals without Narcolepsy. Although joking and laughing are the most typical triggers of cataplexy, episodes that are exclusively triggered by stress or tension or that occur in the context of physical exertion are less likely to represent true cataplexy.

Sleep deprivation from any cause produces daytime sleepiness. Narcolepsy should be diagnosed only if the individual has demonstrated a regular sleep-wake schedule with an adequate amount of nocturnal sleep. Sleep deprivation and irregular sleep schedules may rarely lead to sleep-related hallucinations or sleep paralysis, but not to cataplexy.

The degree of daytime sleepiness may be similar in individuals with Narcolepsy and **Primary Hypersomnia.** Compared with individuals with Narcolepsy, individuals with Primary Hypersomnia generally describe prolonged and less disrupted nocturnal sleep. Daytime sleepiness in Primary Hypersomnia consists of more prolonged, unrefreshing sleep periods, which have less urgency than the sleep "attacks" of Narcolepsy and are less often associated with dreaming. Individuals with Primary Hypersomnia do not manifest cataplexy, sleep-related hallucinations, or sleep paralysis. Nocturnal polysomnography confirms less disrupted sleep and normal REM latency in individuals with Primary Hypersomnia, and the MSLT does not show sleep-onset REM periods.

Individuals with **Breathing-Related Sleep Disorder** often experience excessive sleepiness that is equal in magnitude to that of individuals with Narcolepsy. Furthermore, many individuals with Narcolepsy may develop some degree of sleep apnea. Breathing-Related Sleep Disorder is distinguished from Narcolepsy by a history of loud snoring; breathing pauses that disrupt nocturnal sleep; lengthy, unrefreshing daytime sleep episodes; and the absence of accessory symptoms such as cataplexy. Polysomnography can identify breathing pauses (apneas) in individuals with Breathing-Related Sleep Disorder. If an individual presents with an unambiguous history of Narcolepsy together with confirmatory polysomnographic findings (sleep-onset REM) and also has evidence of Breathing-Related Sleep Disorder during polysom-

nography, both diagnoses can be made. If an individual has sleep-onset REM and sleep apnea activity during polysomnography but does not have the full clinical syndrome of Narcolepsy, then only a diagnosis of Breathing-Related Sleep Disorder should be made.

Individuals with **Hypersomnia Related to Another Mental Disorder** may report excessive sleepiness and intense dreams. In particular, Major Depressive Episodes With Atypical Features and Bipolar Disorder, Most Recent Episode Depressed, often involve an intense need for sleep during the daytime. However, individuals with Mood Disorders typically have prolonged albeit disturbed nocturnal sleep in contrast to the short, fragmented sleep of Narcolepsy. Daytime naps are not refreshing in individuals with Mood Disorders. Furthermore, these individuals do not have the accessory symptoms that are characteristic of Narcolepsy (e.g., cataplexy), although individuals who have Major Depressive Disorder, With Psychotic Features, may complain of hallucinations near sleep and at other times. Polysomnographic studies of individuals with Mood Disorders may reveal short REM latency, but typically not as short as that seen in Narcolepsy. Nocturnal sleep latency is also longer in individuals with Mood Disorders. Finally, daytime testing with the MSLT shows a much lower degree of physiological sleepiness and infrequent sleep-onset REM periods in individuals with Mood Disorders. Thus, the "sleepiness" in these individuals appears to be more a manifestation of psychomotor retardation and anergy.

The **use of, or withdrawal from, substances** (including medications) may produce some symptoms of Narcolepsy. Cholinergic agonists (including anticholinesterase pesticides) can disrupt sleep continuity and enhance REM sleep. Similar effects can result from the abrupt discontinuation of anticholinergic agents, including tricyclic antidepressants. Reserpine and methyldopa can enhance REM sleep and produce sleepiness. Withdrawal from stimulants can produce severe somnolence. A diagnosis of **Substance-Induced Sleep Disorder, Hypersomnia Type,** might be warranted if the symptoms are judged to be due to the direct physiological effects of a substance (see p. 655). Conversely, a diagnosis of Narcolepsy should not be made if the individual is taking or has recently discontinued taking such substances.

Narcolepsy must be distinguished from **Sleep Disorder Due to a General Medical Condition, Hypersomnia Type.** The diagnosis is Sleep Disorder Due to a General Medical Condition when the symptoms are judged to be the direct physiological consequence of a specific general medical condition (e.g., closed head injury or hypothalamic tumor) (see p. 651).

Relationship to the International Classification of Sleep Disorders

The International Classification of Sleep Disorders (ICSD) diagnosis of Narcolepsy includes the same essential features as the DSM-IV diagnosis.

Diagnostic criteria for 347 Narcolepsy

A. Irresistible attacks of refreshing sleep that occur daily over at least 3 months.

B. The presence of one or both of the following:

(1) cataplexy (i.e., brief episodes of sudden bilateral loss of muscle tone, most often in association with intense emotion)

(2) recurrent intrusions of elements of rapid eye movement (REM) sleep into the transition between sleep and wakefulness, as manifested by either hypnopompic or hypnagogic hallucinations or sleep paralysis at the beginning or end of sleep episodes

C. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or another general medical condition.

780.59 Breathing-Related Sleep Disorder

Diagnostic Features

The essential feature of Breathing-Related Sleep Disorder is sleep disruption, leading to excessive sleepiness or, less commonly, to insomnia, that is judged to be due to abnormalities of ventilation during sleep (e.g., sleep apnea or central alveolar hypoventilation) (Criterion A). This sleep disruption must not be better accounted for by a mental disorder and is not due to the direct physiological effects of a substance (including medication) or a general medical condition that produces sleep symptoms through a mechanism other than abnormal breathing (Criterion B).

Excessive sleepiness is the most common presenting complaint of individuals with Breathing-Related Sleep Disorder. Sleepiness results from frequent arousals during nocturnal sleep as the individual attempts to breathe normally. The sleepiness is most evident in relaxing situations, such as when the individual is reading or watching television. The individual's inability to control the sleepiness can be evident in boring meetings or while attending movies, theater, or concerts. When sleepiness is extreme, the person may fall asleep while actively conversing, eating, walking, or driving. Naps tend to be unrefreshing and may be accompanied by a dull headache on awakening. However, there can be considerable variation in the intensity of the sleepiness. The impact of the sleepiness may be minimized by the individual, who may express pride about being able to sleep anywhere at any time.

Insomnia, frequent awakenings, or unrefreshing sleep are less frequent than daytime sleepiness as the presenting complaint in individuals with Breathing-Related Sleep Disorder. Some individuals may complain of difficulty breathing while lying supine or sleeping.

Abnormal respiratory events during sleep in Breathing-Related Sleep Disorder include apneas (episodes of breathing cessation), hypopneas (abnormally slow or shallow respiration), and hypoventilation (abnormal blood oxygen and carbon dioxide

levels). Three forms of Breathing-Related Sleep Disorder have been described: obstructive sleep apnea syndrome, central sleep apnea syndrome, and central alveolar hypoventilation syndrome. An older term, *Pickwickian syndrome*, has been used to describe obese individuals with a combination of obstructive sleep apnea syndrome and waking hypoventilation as well as sleep-related hypoventilation.

Obstructive sleep apnea syndrome is the most common form of Breathing-Related Sleep Disorder. It is characterized by repeated episodes of upper-airway obstruction (apneas and hypopneas) during sleep. The central drive for respiration and respiratory movements in the chest and abdomen are preserved. It usually occurs in overweight individuals and leads to a complaint of excessive sleepiness. Obstructive sleep apnea syndrome is characterized by loud snores or brief gasps that alternate with episodes of silence that usually last 20–30 seconds. Snoring is caused by breathing through a partially obstructed airway. Silent periods are caused by obstructive apneas, with the cessation in breathing caused by complete airway obstruction. Typically the loud snoring has been present for many years, often since childhood, but an increase in its severity may lead the individual to seek evaluation. The snoring is commonly loud enough to disturb the sleep of others in close proximity. The cessation of breathing, sometimes lasting as long as 60–90 seconds and associated with cyanosis, may also be of concern to bedpartners. The termination of the apneic event can be associated with loud "resuscitative" snores, gasps, moans or mumbling, or whole-body movements. The bedpartner may have to move to a separate bed or another room as a result of the affected individual's snoring, gasps, and movements. Most affected individuals are unaware of the loud snoring, breathing difficulty, and frequent arousals. However, some persons, particularly elderly persons, are intensely aware of the sleep disturbance and present with a complaint of frequent awakenings and unrefreshing sleep. Some individuals without frank airway obstruction may demonstrate arousals associated with increased airway resistance (sometimes referred to as *upper airway resistance syndrome* or *respiratory event–related arousals*). These individuals have many clinical characteristics in common with individuals with obstructive sleep apnea syndrome.

Central sleep apnea syndrome is characterized by episodic cessation of ventilation during sleep (apneas and hypopneas) without airway obstruction. Thus, in contrast to obstructive apnea events, central apneas are not associated with continued chest wall and abdominal breathing movements and occur more commonly in elderly persons as a result of cardiac or neurological conditions that affect ventilatory regulation. Individuals most often present with complaints of insomnia due to repeated awakenings, which they may or may not associate with breathing difficulties. Individuals with central sleep apnea may have mild snoring, but it is not a prominent complaint.

The *central alveolar hypoventilation syndrome* is characterized by an impairment in ventilatory control that results in abnormally low arterial oxygen levels further worsened by sleep (hypoventilation without apneas or hypopneas). The lungs in individuals with this disorder have normal mechanical properties. This form most commonly occurs in very overweight individuals and can be associated with a complaint of either excessive sleepiness or insomnia.

Associated Features and Disorders

Associated descriptive features and mental disorders. The individual with Breathing-Related Sleep Disorder may complain of nocturnal chest discomfort, choking, suffocation, or intense anxiety in association with apneic events or hypoventilation. Body movements associated with breathing difficulties can be violent, and individuals with Breathing-Related Sleep Disorder are often described as restless sleepers. Individuals with this disorder typically feel unrefreshed on awakening and may describe feeling more tired in the morning than when they went to sleep. They may also describe sleep drunkenness (i.e., extreme difficulty awakening, confusion, and inappropriate behavior). Severe dryness of the mouth is common and often leads the person to drink water during the night or on awakening in the morning. Nocturia occurs more often with the progression of symptoms. Dull, generalized morning headaches can last for 1–2 hours after awakening.

The sleepiness can lead to memory disturbance, poor concentration, irritability, and personality changes. Mood Disorders (particularly Major Depressive Disorder and Dysthymic Disorder), Anxiety Disorders (particularly Panic Disorder), and dementia are commonly associated with Breathing-Related Sleep Disorder. Individuals can also have reduced libido and erectile ability. Rarely, erectile dysfunction is the presenting complaint of the obstructive sleep apnea syndrome. Children with Breathing-Related Sleep Disorder may have failure to thrive, developmental delay, learning difficulties, poor attention, and hyperactive behavior. Excessive daytime sleepiness can result in injuries (e.g., falling asleep while driving a vehicle) and can also cause severe social and occupational impairment resulting in job loss, marital and family problems, and decreased school performance.

Associated laboratory findings. Each of the major Breathing-Related Sleep Disorder syndromes produces specific abnormalities. In the obstructive sleep apnea syndrome, nocturnal polysomnography shows apneic episodes longer than 10 seconds in duration (usually 20–40 seconds), with rare episodes lasting up to several minutes. Hypopneas are characterized by a reduction of airflow. Both types of events are associated with a reduction in oxyhemoglobin saturation. Generally, more than 10–15 apneas or hypopnias per hour of sleep in the presence of symptoms is considered to be clinically significant. The central sleep apnea syndrome may include Cheyne-Stokes respiration (i.e., a pattern of periodic breathing consisting of an apnea, a 10- to 60-second episode of hyperventilation following the apnea, and a gradual decrease in ventilation culminating in another apnea). In the central alveolar hypoventilation syndrome, periods of decreased respiration lasting up to several minutes occur, with sustained arterial oxygen desaturation and increased carbon dioxide levels. Other features of nocturnal polysomnography in individuals with Breathing-Related Sleep Disorder include short sleep duration, frequent awakenings, increased amounts of stage 1 sleep, and decreased amounts of slow-wave sleep and rapid eye movement (REM) sleep. The arousals that occur at the termination of the apneic and hypoventilation events may be quite brief (several seconds).

Apneas, hypopneas, and hypoventilation may produce other disturbances: oxyhemoglobin desaturation, ECG abnormalities, elevated pulmonary and systemic arterial pressure, and transient arousals as the individual terminates an episode of

breathing disturbance. Cardiac arrhythmias commonly occur during sleep in individuals with Breathing-Related Sleep Disorder and may include sinus arrhythmias, premature ventricular contractions, atrioventricular block, or sinus arrest. Bradycardia followed by tachycardia is commonly seen in association with apneic episodes. Frequent nocturnal awakenings and oxyhemoglobin desaturation can result in excessive sleepiness that may be detected by the Multiple Sleep Latency Test (MSLT) or other tests of daytime sleepiness. Mean sleep latency on the MSLT is often less than 10 minutes and can be less than 5 minutes (normal is 10–20 minutes).

Arterial blood gas measurements while the person is awake are usually normal, but some individuals with severe obstructive sleep apnea syndrome or central alveolar hypoventilation syndrome can have waking hypoxemia or hypercarbia. Cephalometric X rays, magnetic resonance imaging (MRI), computed tomography (CT), and fiber-optic endoscopy can show obstruction of the upper airway. Cardiac testing may show evidence of impaired right ventricular function. Individuals may also have elevated hemoglobin or hematocrit values due to repeated nocturnal hypoxemia. Polysomnographic findings in children differ from those in adults in that most children demonstrate labored breathing, partial obstructive hypoventilation with cyclical desaturations, hypercapnia, paradoxical movements, and snoring.

Associated physical examination findings and general medical conditions. The majority of individuals with the obstructive sleep apnea syndrome and the central alveolar hypoventilation syndrome are overweight and notice an increase in the severity of symptoms with increasing body weight. Upper-airway narrowing can occur due to excessive bulk of soft tissues. In particular, individuals with larger neck sizes (e.g., neck circumference greater than 17 inches in men and greater than 16 inches in women) are at higher risk for obstructive sleep apnea. Obstructive sleep apnea syndrome occurring in individuals of normal or below-normal body weight suggests upper-airway obstruction due to definable, localized structural abnormality, such as a maxillomandibular malformation or adenotonsillar enlargement. Nasal airway obstruction may also be present. Individuals may have noisy breathing even while awake. Gastroesophageal reflux with severe "heartburn" pain may occur in the obstructive sleep apnea syndrome in association with the effort to reestablish breathing during sleep. Individuals with central sleep apnea syndrome less frequently are overweight or have demonstrable upper-airway obstructions.

Systemic hypertension with elevated diastolic pressure is commonly associated with Breathing-Related Sleep Disorder. Some individuals, particularly those with chronic obstructive pulmonary disease or alveolar hypoventilation, have continuously low oxygen saturation values during sleep and are predisposed to developing pulmonary hypertension and associated right-sided cardiac failure (cor pulmonale), hepatic congestion, and ankle edema.

Individuals with Breathing-Related Sleep Disorder may have an underlying abnormality in the neurological control of the upper-airway musculature or ventilation during sleep. Disorders affecting neurological control of ventilation usually manifest as the central sleep apnea syndrome. Some individuals with neurological conditions have a specific lesion affecting the control of pharyngeal muscles, which may lead to the obstructive sleep apnea syndrome.

Breathing-Related Sleep Disorder can be associated with systemic general medical

or neurological conditions. For instance, obstructive sleep apnea may result from tongue enlargement due to acromegaly, lingual thyroid tissue or cysts, or vocal cord paralysis as seen in Shy-Drager syndrome. Impaired cardiac function due to reduced cardiac output can result in central sleep apnea, as can neurological conditions that affect the brain stem control of respiration, such as syringobulbia or brain stem tumors.

Specific Age and Gender Features

In young children, the signs and symptoms of Breathing-Related Sleep Disorder (almost exclusively the obstructive sleep apnea syndrome) are more subtle than those in adults and the diagnosis is more difficult to establish. In children, polysomnography is useful in confirming the diagnosis. Snoring, which is characteristic of adult obstructive sleep apnea syndrome, might not be present. Agitated arousals and unusual sleep postures, such as sleeping on the hands and knees, commonly occur. Nocturnal enuresis is also common and should raise the suspicion of obstructive sleep apnea syndrome if it recurs in a child who was previously dry at night. Children may also manifest excessive daytime sleepiness, although this is not as common or pronounced as in adults. Daytime mouth breathing, difficulty in swallowing, and poor speech articulation are also common features in children. In children younger than 5 years, nighttime symptoms such as observed apneas or labored breathing are more often the presenting symptoms. In children over the age of 5, daytime symptoms such as sleepiness and behavioral problems, attention and learning difficulties, and morning headaches are more often the focus of concern. On physical examination, pectus excavatum and rib flaring can be seen. If associated with adenotonsillar enlargement (the most common cause of obstructive sleep apnea in children), typical "adenoid facies" can be seen with a dull expression, periorbital edema, and mouth breathing.

The obstructive sleep apnea syndrome is most common in middle-aged, overweight males and prepubertal children with enlarged tonsils. Aging leads to an increase in the frequency of both obstructive and central apnea events, even among asymptomatic healthy individuals. Because some degree of apnea may be normative with aging, polysomnographic results must be interpreted within this context. On the other hand, significant clinical symptoms of insomnia and hypersomnia should be investigated regardless of the individual's age, and a diagnosis of Breathing-Related Sleep Disorder should be made if a breathing disturbance best explains the symptoms.

In adults, the male-to-female ratio of obstructive sleep apnea syndrome ranges from 2:1 to 4:1. There is no sex difference among prepubertal children. In adults, central apneic events appear to be more prevalent in males than in females, although this difference is less apparent after menopause.

Prevalence

The prevalence of Breathing-Related Sleep Disorder associated with obstructive sleep apnea is estimated to be approximately 1%–10% in the adult population but may be higher in elderly individuals. The prevalence of Breathing-Related Sleep Disorder also varies considerably as a function of the threshold for the frequency of apnea

events. The prevalence of central sleep apnea syndrome is not precisely known but is estimated to be 10% of the rate of obstructive sleep apnea syndrome.

Course

The obstructive sleep apnea syndrome can occur at any age, but most individuals present for evaluation when they are between ages 40 and 60 years (with females more likely to develop obstructive sleep apnea after menopause). Central sleep apnea is more commonly seen in elderly individuals with central nervous system or cardiac disease. The central alveolar hypoventilation syndrome and central sleep apnea syndrome can develop at any age.

Breathing-Related Sleep Disorder usually has an insidious onset, gradual progression, and chronic course. Most often, the disorder will have been present for years by the time it is diagnosed. Spontaneous resolution of the obstructive sleep apnea syndrome has been reported with weight loss, but usually the course is progressive and can ultimately lead to premature death due to cardiovascular disease or arrhythmia. The central sleep apnea syndrome also has a chronic unremitting course, although management of underlying medical conditions may improve the breathing disturbance. Adults with the central alveolar hypoventilation syndrome have a slowly progressive course.

Familial Pattern

A familial tendency for obstructive sleep apnea syndrome has been described.

Differential Diagnosis

Breathing-Relating Sleep Disorder must be differentiated from other causes of sleepiness, such as Narcolepsy, Primary Hypersomnia, and Circadian Rhythm Sleep Disorder. Breathing-Related Sleep Disorder can be differentiated from **Narcolepsy** by the absence of cataplexy, sleep-related hallucinations, and sleep paralysis and by the presence of loud snoring, gasping during sleep, or observed apneas or shallow breathing in sleep. Daytime sleep episodes in Narcolepsy are characteristically shorter, more refreshing, and more often associated with dreaming. Breathing-Related Sleep Disorder shows characteristic apneas or hypoventilation during nocturnal polysomnographic studies, and Narcolepsy results in multiple sleep-onset REM periods during the MSLT. Some individuals have concurrent Narcolepsy and Breathing-Related Sleep Disorder. Breathing-Related Sleep Disorder may be distinguished from **Primary Hypersomnia** and **Circadian Rhythm Sleep Disorder** based on the presence of clinical or laboratory findings of obstructive sleep apnea, central sleep apnea, or central alveolar hypoventilation syndromes. Definitive differential diagnosis between Primary Hypersomnia and Breathing-Related Sleep Disorder may require polysomnographic studies.

Hypersomnia related to a Major Depressive Episode can be distinguished from Breathing-Related Sleep Disorder by the presence or absence of other characteristic symptoms (e.g., depressed mood and loss of interest in a Major Depressive Episode and snoring and gasping during sleep in Breathing-Related Sleep Disorder).

Individuals with Breathing-Related Sleep Disorder must also be differentiated from otherwise **asymptomatic adults who snore.** This differentiation can be made based on the presenting complaint of insomnia or hypersomnia, the greater intensity of snoring, and the presence of the characteristic history, signs, and symptoms of Breathing-Related Sleep Disorder. For individuals complaining of insomnia, **Primary Insomnia** can be differentiated from Breathing-Related Sleep Disorder by the absence of complaints (or reports from bedpartners) of difficulty breathing during sleep and the absence of the history, signs, and symptoms characteristic of Breathing-Related Sleep Disorder.

Nocturnal Panic Attacks may include symptoms of gasping or choking during sleep that may be difficult to distinguish clinically from Breathing-Related Sleep Disorder. However, the lower frequency of episodes, intense autonomic arousal, and the lack of excessive sleepiness differentiates nocturnal Panic Attacks from Breathing-Related Sleep Disorder. Polysomnography in individuals with nocturnal Panic Attacks does not reveal the typical pattern of apneas, hypoventilation, or oxygen desaturation characteristic of Breathing-Related Sleep Disorder.

Attention-Deficit/Hyperactivity Disorder in children may include symptoms of inattention, academic impairment, and hyperactivity, all of which may also be symptoms of childhood sleep apnea. The presence of other symptoms and signs of childhood sleep apnea (e.g., labored breathing or snoring during sleep and adenotonsillar hypertrophy) would suggest the presence of a Breathing-Related Sleep Disorder.

The diagnosis of Breathing-Related Sleep Disorder is appropriate in the presence of a **general medical condition** that causes insomnia or hypersomnia through the mechanism of impaired ventilation during sleep. For example, an individual with tonsillar hypertrophy who has sleep difficulty related to snoring and obstructive sleep apneas should receive a diagnosis of Breathing-Related Sleep Disorder on Axis I and tonsillar hypertrophy on Axis III. In contrast, Sleep Disorder Due to a General Medical Condition is appropriate if a general medical or neurological condition causes sleep-related symptoms through a mechanism other than breathing disturbance. For instance, individuals with arthritis or renal impairment may complain of insomnia or hypersomnia, but this does not result from breathing impairment during sleep.

The **use of, or withdrawal from, substances** (including medications) can produce insomnia or hypersomnia similar to that in Breathing-Related Sleep Disorder. A careful history is usually sufficient to identify the relevant substance, and follow-up shows improvement of the sleep disturbance after discontinuation of the substance. In other cases, the use of a substance (e.g., alcohol, barbiturates, benzodiazepines, or tobacco) has been shown to be associated with Breathing-Related Sleep Disorder. An individual with symptoms and signs consistent with Breathing-Related Sleep Disorder should receive that diagnosis, even in the presence of concurrent substance use that is exacerbating the condition.

Relationship to the International Classification of Sleep Disorders

Breathing-Related Sleep Disorder is identified as three more specific syndromes in the International Classification of Sleep Disorders (ICSD): Obstructive Sleep Apnea Syndrome, Central Sleep Apnea Syndrome, and Central Alveolar Hypoventilation Syndrome.

Diagnostic criteria for 780.59 Breathing-Related Sleep Disorder

A. Sleep disruption, leading to excessive sleepiness or insomnia, that is judged to be due to a sleep-related breathing condition (e.g., obstructive or central sleep apnea syndrome or central alveolar hypoventilation syndrome).

B. The disturbance is not better accounted for by another mental disorder and is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or another general medical condition (other than a breathing-related disorder).

Coding note: Also code sleep-related breathing disorder on Axis III.

307.45 Circadian Rhythm Sleep Disorder (*formerly* Sleep-Wake Schedule Disorder)

Diagnostic Features

The essential feature of Circadian Rhythm Sleep Disorder is a persistent or recurrent pattern of sleep disruption that results from altered function of the circadian timing system or from a mismatch between the individual's endogenous circadian sleep-wake system and exogenous demands regarding the timing and duration of sleep (Criterion A). In contrast to other primary Sleep Disorders, Circadian Rhythm Sleep Disorder does not result from dysfunction of the mechanisms generating sleep and wakefulness per se. As a result of the circadian mismatch, individuals with this disorder may complain of insomnia at certain times during the 24-hour day and excessive sleepiness at other times, with resulting impairment in social, occupational, or other important areas of functioning or marked subjective distress (Criterion B). The sleep problems are not better accounted for by other Sleep Disorders or other mental disorders (Criterion C) and are not due to the direct physiological effects of a substance or a general medical condition (Criterion D).

The diagnosis of Circadian Rhythm Sleep Disorder should be reserved for those presentations in which the individual has significant social or occupational impairment or marked distress related to the sleep disturbance. Individuals vary widely in their ability to adapt to circadian changes and requirements. Many, if not most, individuals with circadian-related symptoms of sleep disturbance do not seek treatment and do not have symptoms of sufficient severity to warrant a diagnosis. Those who present for evaluation because of this disorder are most often troubled by the severity or persistence of their symptoms. For example, it is not unusual for shift workers to present for evaluation after falling asleep while on the job or while driving.

The diagnosis of Circadian Rhythm Sleep Disorder rests primarily on the clinical history, including the pattern of work, sleep, naps, and "free time." The history should also examine past attempts at coping with symptoms, such as attempts at ad-

vancing the sleep-wake schedule in Delayed Sleep Phase Type. Prospective sleep-wake diaries or sleep charts are often a useful adjunct to diagnosis.

Subtypes

Delayed Sleep Phase Type. This type of Circadian Rhythm Sleep Disorder is characterized by a delay of circadian rhythms, including the sleep-wake cycle, relative to the demands of society. Measurement of endogenous circadian rhythms (e.g., core body temperature, plasma melatonin levels) during the individual's usual (i.e., delayed) sleep-wake schedule reflects this delay. Individuals with this subtype are hypothesized to have an abnormally diminished ability to phase-advance sleep-wake hours (i.e., to move sleep and wakefulness to earlier clock times) or an alteration in the usual alignment of sleep with other circadian rhythms. As a result, these individuals are "locked in" to habitually late sleep hours and have great difficulty shifting these sleep hours forward to an earlier time. The circadian phase of sleep is stable: individuals will fall asleep and awaken at consistent, albeit delayed, times when left to their own schedule (e.g., on weekends or vacations). Affected individuals complain of difficulty falling asleep at socially acceptable hours, but once sleep is initiated, it is normal. There is concomitant difficulty awakening at socially acceptable hours (e.g., multiple alarm clocks are often unable to arouse the individual). Many individuals with this disorder will be chronically sleep deprived as a result of the need to awaken for social and occupational obligations in the morning. Sleepiness during the desired wake period may result.

Jet Lag Type. In this type of Circadian Rhythm Sleep Disorder, the endogenous circadian sleep-wake cycle is normal and the disturbance arises from conflict between the pattern of sleep and wakefulness generated by the circadian system and the pattern of sleep and wakefulness required by a new time zone. Individuals with this type complain of a mismatch between desired and required hours of sleep and wakefulness. The severity of symptoms is proportional to the number of time zones traveled, with maximal difficulties often noted after traveling through eight or more time zones in less than 24 hours. Eastward travel (advancing sleep-wake hours) is typically more difficult for most individuals to tolerate than westward travel (delaying sleep-wake hours).

Shift Work Type. In this type of Circadian Rhythm Sleep Disorder, the endogenous circadian sleep-wake cycle is normal and the disturbance arises from conflict between the pattern of sleep and wakefulness generated by the circadian system and the desired pattern of sleep and wakefulness required by shift work. Night-shift schedules (with shifting back to a day schedule on days off) and rotating-shift schedules are the most disruptive because they force sleep and wakefulness into aberrant circadian positions and prevent any consistent adjustment. Workers on these shifts typically have a shorter sleep duration and more frequent disturbances in sleep continuity than morning and afternoon workers. Conversely, these individuals may feel sleepy or fall asleep during the desired wake period, that is, in the middle of the night work shift. Work schedules that involve slowly rotating shifts or rotations to progressively

earlier shifts (i.e., nights–afternoons–days) are also associated with higher degrees of sleep disturbance and other complaints than other types of rotating-shift schedules. The circadian mismatch of the Shift Work Type is further exacerbated by insufficient sleep time, social and family demands, alcohol use, and environmental disturbances (e.g., telephone, traffic noise) during intended sleep times.

Unspecified Type. This type of Circadian Rhythm Sleep Disorder should be indicated if another pattern of circadian sleep disturbance (e.g., advanced sleep phase, non-24-hour sleep-wake pattern, or irregular sleep-wake pattern) is present. An "advanced sleep phase pattern" is the analog of Delayed Sleep Phase Type, but in the opposite direction: individuals complain of an inability to stay awake in the evening and spontaneous awakening in the early morning hours. "Non-24-hour sleep-wake pattern" denotes a free-running cycle: the sleep-wake schedule follows the endogenous circadian rhythm period of slightly over 24 hours despite the presence of 24-hour time cues in the environment. In contrast to the stable sleep-wake pattern of the Delayed or advanced sleep phase types, these individuals' sleep-wake schedules become progressively delayed relative to the 24-hour clock, resulting in a changing sleep-wake pattern and changing sleep-wake complaints over successive days (e.g., several days of sleep-onset insomnia followed by days of daytime sleepiness followed by days of difficulty staying awake in the evening). "Irregular sleep-wake pattern" indicates the absence of an identifiable pattern of sleep and wakefulness.

Associated Features and Disorders

Associated descriptive features and mental disorders. In Delayed Sleep Phase Type, individuals frequently go to bed later and wake up later on weekends or during vacations, with a reduction in sleep-onset difficulties and difficulty awakening. They will typically give many examples of school, work, and social difficulties arising from their difficulty awakening at socially desired times. If awakened earlier than the time dictated by the circadian timekeeping system, the individual may demonstrate "sleep drunkenness" (i.e., extreme difficulty awakening, confusion, and inappropriate behavior). Performance often also follows a delayed phase, with peak efficiency occurring in late-evening hours.

Jet Lag Type may be more common in individuals who are "morning larks." It is often accompanied by other symptoms such as impaired concentration and memory, impaired coordination, weakness, lightheadedness, headache, fatigue, malaise, decreased appetite, and indigestion. These symptoms may relate not only to circadian mismatch but also to other travel conditions such as sleep deprivation, alcohol and caffeine use, and decreased ambient air pressure in airplanes. Performance is often impaired, following the pattern that would be predicted by the underlying endogenous circadian rhythms. Shift Work Type may also be more common in individuals who are "morning types." Concentration and attention, performance, and alertness are often impaired during desired waking hours, following the pattern that would be predicted by the underlying endogenous circadian rhythms. Reduced quality of life and dysfunction in occupational, family, and social roles are often observed in shift

workers, particularly those who have sleep difficulties. Shift work is a risk factor for sleepiness-related work and motor vehicle accidents.

The non-24-hour sleep-wake pattern has been described primarily in blind individuals, particularly those with no light perception (e.g., from retrolental fibroplasia or surgical enucleation) as opposed to those with some degree of conscious light perception. Napping and regularly recurring insomnia occur when the individual's endogenous circadian rhythms (which are slightly longer than 24 hours) are out of phase with the light-dark cycle and socially appropriate sleep-wake hours.

Individuals with any Circadian Rhythm Sleep Disorder may use increased amounts of alcohol, sedative-hypnotic, or stimulants in an attempt to control their inappropriately phased sleep-wake tendencies. The use of these substances may in turn exacerbate the Circadian Rhythm Sleep Disorder. Delayed Sleep Phase Type has been associated with schizoid, schizotypal, and avoidant personality features, particularly in adolescents, as well as with depressive symptoms and Depressive Disorders. "Non-24-hour sleep-wake pattern" and "irregular sleep-wake pattern" have also been associated with these same features. Jet Lag and Shift Work Types may precipitate or exacerbate a Manic or Major Depressive Episode or an episode of a Psychotic Disorder. Shift work is also associated with depressive symptoms.

Associated laboratory findings. Sleep studies yield different results depending on what time they are performed. For individuals with Delayed Sleep Phase Type, studies conducted at the preferred sleep times will be essentially normal for age. However, when studied at socially normal sleep times, these individuals have prolonged sleep latency, reduced sleep efficiency (due mainly to sleep-onset difficulties), short sleep duration, and, in some individuals, moderately short REM sleep latency. Laboratory procedures designed to measure the phase of the endogenous circadian pacemaker (e.g., core body temperature, plasma melatonin levels) reveal the expected phase delay in the timing of acrophase (peak time) and nadir when individuals are studied during their usual sleep-wake times. In addition, awakening time may be delayed relative to other circadian rhythms.

When studied during their habitual workweek sleep hours, individuals with Shift Work Type usually have normal or short sleep latency, reduced sleep duration, and more frequent sleep continuity disturbances compared with age-matched individuals with "normal" nocturnal sleep patterns. There may be a specific reduction in stage 2, stages 3 and 4, and REM sleep in many cases. Polysomnographic patterns in shift workers have been shown to remain stable over intervals of 2 years, suggesting neither adaptation nor worsening. Measures of physiological sleepiness, such as the Multiple Sleep Latency Test (MSLT), show a high degree of sleepiness during desired wake times (e.g., during the night shift). After a period of adjustment to a normal diurnal schedule, these individuals have normal nocturnal sleep and normal levels of daytime sleepiness. When studied on their usual shift-work schedules in their usual environment, shift workers demonstrate changes in the overt timing of their circadian rhythms relative to normal diurnal patterns. However, these changes seldom if ever result in a complete nocturnal orientation. Interventions such as bright light can shift endogenous circadian rhythms into phase with the night shift, but this does not necessarily improve subjective complaints or performance. Night shift work may be associated with increases in triglyceride or cholesterol levels.

Laboratory studies of simulated jet lag demonstrate prolonged sleep latency, impaired sleep efficiency, reductions in REM sleep, and minor reductions in slow-wave sleep. These features recover toward baseline values over 1–2 weeks and are more severe with simulated eastward travel (i.e., advanced sleep hours) than with simulated westward travel (i.e., delayed sleep hours). Other laboratory measures, including circadian rhythms of melatonin, core body temperature, alertness, and performance, also take several days or weeks to adjust following simulated jet lag. Non-24-hour sleep-wake pattern in blind individuals is often characterized by "free-running" circadian rhythms of core body temperature, melatonin secretion, and sleep propensity. In other words, these rhythms have a period of slightly longer than 24 hours, similar to those of sighted individuals deprived of all time cues in experimental settings. Individuals with "advanced sleep phase pattern," as expected, show earlier timing of endogenous circadian rhythms, as well as a shortening of the endogenous circadian rhythm period.

Associated physical examination findings and general medical conditions. No specific physical findings are described for Circadian Rhythm Sleep Disorder. Shift workers may appear haggard or sleepy and may have an excess of gastrointestinal disturbances, including gastritis and peptic ulcer disease. The roles of caffeine and alcohol consumption and altered eating patterns have not been fully evaluated in these cases. Shift work has been associated with risk factors for cardiovascular disease, such as hypertension, "nondipping" 24-hour blood pressure patterns, increased atherogenic lipids, and abnormal electrocardiographic measures (prolonged QT_c interval). It may also be associated with a slightly increased risk for actual cardiovascular disease, although not all studies have found this. Non-24-hour sleep-wake pattern often occurs in blind individuals, particularly those with no light perception. Circadian Rhythm Sleep Disorder may exacerbate preexisting general medical conditions.

Specific Age Features

The onset of Delayed Sleep Phase Type most often occurs between late childhood and early adulthood. Shift work and jet lag symptoms are often reported to be more severe, or more easily induced in laboratory settings, in late-middle-aged and elderly individuals compared with young adults. Older adults also have more severe polysomnographic sleep disturbances following simulated jet lag in the laboratory, but their circadian rhythms appear to adjust at the same rate as younger adults. "Advanced sleep phase pattern" also increases with age. These findings may result from age-related deterioration in nocturnal sleep and shortening of the endogenous circadian period.

Prevalence

The prevalence for any of the types of Circadian Rhythm Sleep Disorder has not been well established. Prevalence figures for the Delayed Sleep Phase Type from population surveys have varied widely, ranging from 0.1% to 4% in adults and up to 7% in adolescents. Up to 60% of night shift workers may have Shift Work Type.

Course

Delayed Sleep Phase Type typically begins during adolescence and may follow a psychosocial stressor. Without intervention, Delayed Sleep Phase Type typically lasts for years or decades but may "correct" itself given the tendency for endogenous circadian rhythm phase to advance with age. Treatment can often normalize sleep hours at least temporarily, but there is a persistent vulnerability for delayed sleep hours and other symptoms.

Shift Work Type typically persists for as long as the individual works that particular schedule. Reversal of symptoms generally occurs within 2 weeks of a return to a normal diurnal sleep-wake schedule.

Experimental and field data concerning jet lag indicate that it takes approximately 1 day per time zone traveled for the circadian system to resynchronize itself to the new local time. Different circadian rhythms (such as core body temperature, hormonal level, alertness, and sleep patterns) may readjust at different rates.

Familial Pattern

A family history may be present in up to 40% of individuals with Delayed Sleep Phase Type. A familial form of Advanced Sleep Phase Type, segregating as an autosomal dominant trait with high penetrance, has been identified.

Differential Diagnosis

Circadian Rhythm Sleep Disorder must be distinguished from **normal patterns of sleep** and **normal adjustments following a change in schedule.** The key to such distinctions lies in the persistence of the disturbance and the presence and degree of social or occupational impairment. For instance, many adolescents and young adults maintain delayed sleep-wake schedules, but without distress or interference with school or work routines. Likewise, many individuals characterize themselves as either "night owls" or "morning larks," because of their preference for either late or early sleep schedules. These tendencies in themselves do not warrant a diagnosis of Delayed Sleep Phase Type or "advanced sleep phase pattern." A diagnosis would be made only in individuals who persistently experience clinically significant distress or impairment and who have difficulty changing their sleep-wake pattern. Similarly, almost anyone who travels across time zones will experience transient sleep disruption. The diagnosis of the Jet Lag Type should be reserved for individuals with associated severe sleep disturbances and work disruption.

Delayed Sleep Phase Type must be differentiated from **volitional patterns of delayed sleep hours.** Some individuals who voluntarily delay sleep onset to participate in social or work activities may complain of difficulty awakening. When permitted to do so, these individuals fall asleep readily at earlier times and, after a period of recovery sleep, have no significant difficulty awakening in the morning. In such cases, the primary problem is sleep deprivation rather than a Circadian Rhythm Sleep Disorder. Other individuals (particularly children and adolescents) may volitionally shift sleep hours to avoid school or family demands. The pattern of difficulty awakening vanishes when desired activities are scheduled in the morning hours. In a similar

way, younger children involved in limit-setting battles with parents may present as having Delayed Sleep Phase Type.

Jet Lag and Shift Work Types must be distinguished mainly from other primary Sleep Disorders, such as **Primary Insomnia** and **Primary Hypersomnia.** The history of jet lag or shift work, with undisturbed sleep on other schedules, usually provides sufficient evidence to exclude these other disorders. In some cases, other primary Sleep Disorders, such as **Breathing-Related Sleep Disorder** or **periodic limb movements disorder,** may complicate Shift Work or Jet Lag Types. This possibility should be suspected when reversion to a normal diurnal schedule does not provide relief from sleep-related symptoms. Other types of Circadian Rhythm Sleep Disorder, such as "non-24-hour sleep-wake pattern" and "irregular sleep-wake pattern," are distinguished from the Delayed Sleep Phase Type by the stable pattern of delayed sleep-wake hours characteristic of the latter.

Patterns of delayed or advanced sleep that occur exclusively during another mental disorder are not diagnosed separately. For instance, an individual with Major Depressive Disorder may have delayed sleep hours similar to those in Delayed Sleep Phase Type, but if this sleep pattern occurs only during the Major Depressive Episode, an additional diagnosis of Circadian Rhythm Sleep Disorder would not be warranted. Likewise, an individual experiencing an acute exacerbation of Schizophrenia may have a very irregular sleep-wake pattern, but if this sleep pattern is only associated with the exacerbation, no additional diagnosis of Circadian Rhythm Sleep Disorder would be made.

Substances (including medications) can cause delayed sleep onset or awakening in the morning. For instance, consumption of caffeine or nicotine in the evening may delay sleep onset, and the use of hypnotic medications in the middle of the night may delay the time of awakening. A diagnosis of **Substance-Induced Sleep Disorder** may be considered if the sleep disturbance is judged to be a direct physiological consequence of regular substance use and warrants independent clinical attention (see p. 655). General medical conditions rarely cause fixed delays or advances of the sleep-wake schedule and typically pose no difficulty in differential diagnosis.

Relationship to the International Classification of Sleep Disorders

The International Classification of Sleep Disorders (ICSD) includes categories for Delayed Sleep Phase Syndrome, Shift Work Sleep Disorder and Time Zone Change (Jet Lag) Syndrome, and specific categories for three other Circadian Rhythm Sleep Disorders (Irregular Sleep-Wake Pattern, Advanced Sleep Phase Syndrome, and Non-24-Hour Sleep-Wake Syndrome).

Diagnostic criteria for 307.45 Circadian Rhythm Sleep Disorder

A. A persistent or recurrent pattern of sleep disruption leading to excessive sleepiness or insomnia that is due to a mismatch between the sleep-wake schedule required by a person's environment and his or her circadian sleep-wake pattern.

B. The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The disturbance does not occur exclusively during the course of another Sleep Disorder or other mental disorder.

D. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Specify type:

Delayed Sleep Phase Type: a persistent pattern of late sleep onset and late awakening times, with an inability to fall asleep and awaken at a desired earlier time

Jet Lag Type: sleepiness and alertness that occur at an inappropriate time of day relative to local time, occurring after repeated travel across more than one time zone

Shift Work Type: insomnia during the major sleep period or excessive sleepiness during the major awake period associated with night shift work or frequently changing shift work

Unspecified Type

307.47 Dyssomnia Not Otherwise Specified

The Dyssomnia Not Otherwise Specified category is for insomnias, hypersomnias, or circadian rhythm disturbances that do not meet criteria for any specific Dyssomnia. Examples include

1. Complaints of clinically significant insomnia or hypersomnia that are attributable to environmental factors (e.g., noise, light, frequent interruptions).
2. Excessive sleepiness that is attributable to ongoing sleep deprivation.
3. "Restless legs syndrome": This syndrome is characterized by a desire to move the legs or arms, associated with uncomfortable sensations typically described as creeping, crawling, tingling, burning, or itching. Frequent movements of the limbs occur in an effort to relieve the uncomfortable sensations. Symptoms are worse when the individual is at rest and in the evening or night, and they are relieved temporarily by movement. The uncomfortable sensations and limb movements can delay sleep onset, awaken the individual from sleep, and lead to daytime sleepiness or fatigue. Sleep studies demonstrate involuntary periodic limb movements during sleep in a majority of individuals with restless legs syndrome. A minority of individuals have evidence of anemia or reduced serum

iron stores. Peripheral nerve electrophysiological studies and gross brain morphology are usually normal. Restless legs syndrome can occur in an idiopathic form, or it can be associated with general medical or neurological conditions, including normal pregnancy, renal failure, rheumatoid arthritis, peripheral vascular disease, or peripheral nerve dysfunction. Phenomenologically, the two forms are indistinguishable. The onset of restless legs syndrome is typically in the second or third decade, although up to 20% of individuals with this syndrome may have symptoms before age 10. The prevalence of restless legs syndrome is between 2% and 10% in the general population and as high as 30% in general medical populations. Prevalence increases with age and is equal in males and females. Course is marked by stability or worsening of symptoms with age. There is a positive family history in 50%–90% of individuals. The major differential diagnoses include medication-induced akathisia, peripheral neuropathy, and nocturnal leg cramps. Worsening at night and periodic limb movements are more common in restless legs syndrome than in medication-induced akathisia or peripheral neuropathy. Unlike restless legs syndrome, nocturnal leg cramps do not present with the desire to move the limbs nor are there frequent limb movements.

4. Periodic limb movements: Periodic limb movements are repeated low-amplitude brief limb jerks, particularly in the lower extremities. These movements begin near sleep onset and decrease during stage 3 or 4 non–rapid eye movement (NREM) and rapid eye movement (REM) sleep. Movements usually occur rhythmically every 20–60 seconds and are associated with repeated, brief arousals. Individuals are often unaware of the actual movements, but may complain of insomnia, frequent awakenings, or daytime sleepiness if the number of movements is very large. Individuals may have considerable variability in the number of periodic limb movements from night to night. Periodic limb movements occur in the majority of individuals with restless legs syndrome, but they may also occur without the other symptoms of restless legs syndrome. Individuals with normal pregnancy or with conditions such as renal failure, congestive heart failure, and Posttraumatic Stress Disorder may also develop periodic limb movements. Although typical age at onset and prevalence in the general population are unknown, periodic limb movements increase with age and may occur in more than one-third of individuals over age 65. Men are more commonly affected than women.
5. Situations in which the clinician has concluded that a Dyssomnia is present but is unable to determine whether it is primary, due to a general medical condition, or substance induced.

Parasomnias

Parasomnias are disorders characterized by abnormal behavioral or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions. Unlike dyssomnias, parasomnias do not involve abnormalities of the mechanisms generating sleep-wake states, nor of the timing of sleep and wakefulness. Rather, parasomnias represent the activation of physiological systems at inappropriate times during the sleep-wake cycle. In particular, these disorders involve activation of the autonomic nervous system, motor system, or cognitive processes during

sleep or sleep-wake transitions. Different parasomnias occur at different times during sleep, and specific parasomnias often occur during specific sleep stages. Individuals with parasomnias usually present with complaints of unusual behavior during sleep rather than complaints of insomnia or excessive daytime sleepiness. This section includes Nightmare Disorder, Sleep Terror Disorder, Sleepwalking Disorder, and Parasomnia Not Otherwise Specified.

307.47 Nightmare Disorder (*formerly* Dream Anxiety Disorder)

Diagnostic Features

The essential feature of Nightmare Disorder is the repeated occurrence of frightening dreams that lead to awakenings from sleep (Criterion A). The individual becomes fully alert on awakening (Criterion B). The frightening dreams or sleep interruptions resulting from the awakenings cause the individual significant distress or result in social or occupational dysfunction (Criterion C). This disorder is not diagnosed if the nightmares occur exclusively during the course of another mental disorder or are due to the direct physiological effects of a substance (e.g., a drug of abuse or a medication) or a general medical condition (Criterion D).

Nightmares typically occur in a lengthy, elaborate dream sequence that is highly anxiety provoking or terrifying. Dream content most often focuses on imminent physical danger to the individual (e.g., pursuit, attack, injury). In other cases, the perceived danger may be more subtle, involving personal failure or embarrassment. Nightmares that occur after traumatic experiences may replicate the original dangerous or threatening situation, but most nightmares do not recount actual events. On awakening, individuals with this disorder can describe the dream sequence and content in detail. Individuals may report multiple nightmares within a given night, often with a recurrent theme. Nightmares arise almost exclusively during rapid eye movement (REM) sleep. Because REM episodes occur periodically throughout nocturnal sleep (approximately every 90–110 minutes), nightmares may also occur at any time during the sleep episode. However, because REM sleep periods typically become longer and dreaming more intense in the second half of the night, nightmares are also more likely to occur later in the night.

Nightmares usually terminate with an awakening that is associated with a rapid return of full alertness and a lingering sense of fear or anxiety. These factors often lead to difficulty returning to sleep. Nightmare Disorder causes significant subjective distress more often than it causes demonstrable social or occupational impairment. However, if nocturnal awakenings are frequent, or if the individual avoids sleeping because of fear of nightmares, the individual may experience excessive sleepiness, poor concentration, depression, anxiety, or irritability that can disrupt daytime functioning.

Associated Features and Disorders

Associated descriptive features and mental disorders. In individuals with Nightmare Disorder, mild autonomic arousal (e.g., sweating, tachycardia, tachypnea) may be evident on awakening. Individuals who have had frequent nightmares since childhood tend to show elevated rates of general psychopathology on symptom measures. Depressive and anxiety symptoms that do not meet criteria for a specific diagnosis are common among individuals with Nightmare Disorder. Body movements and vocalization are not characteristic of Nightmare Disorder because of the loss of skeletal muscle tone that normally occurs during REM sleep. When talking, screaming, or striking out do occur, these are most likely to appear as brief phenomena that terminate a nightmare. These behaviors are also more likely to occur in the nightmares that accompany Posttraumatic Stress Disorder, because these nightmares may occur during non–rapid eye movement (NREM) sleep.

Associated laboratory findings. Polysomnographic studies demonstrate abrupt awakenings from REM sleep that correspond to the individual's report of nightmares. These awakenings usually occur during the second half of the night. In most cases, the REM sleep episode will have lasted for more than 10 minutes and may include a greater-than-average number of eye movements. Heart rate and respiratory rate may increase or show increased variability before the awakening. Nightmares following traumatic events (e.g., in individuals with Posttraumatic Stress Disorder) may arise during NREM sleep, particularly stage 2, as well as during REM sleep. Other polysomnographic features, including sleep continuity and sleep architecture, are not characteristically abnormal in Nightmare Disorder.

Specific Culture, Age, and Gender Features

The significance attributed to nightmares may vary with cultural background. For instance, some cultures may relate nightmares to spiritual or supernatural phenomena, whereas others may view nightmares as indicators of mental or physical disturbance. Because nightmares frequently occur during childhood, this diagnosis should not be given unless there is persistent significant distress or impairment that warrants independent clinical attention. Nightmare Disorder is most likely to appear in children exposed to severe psychosocial stressors. Although specific dream content may reflect the age of the individual having the nightmares, the essential features of the disorder are the same across age groups. Females report having nightmares more often than do men, at a ratio of approximately 2:1 to 4:1. It is not clear to what extent this difference reflects a true discrepancy in the number of nightmares as opposed to a variance in reporting.

Prevalence

Between 10% and 50% of children ages 3–5 years have nightmares of sufficient intensity to disturb their parents. In the adult population, as many as 50% of individuals may report at least an occasional nightmare. In young adults, at least 3% report having nightmares frequently or always. However, the actual prevalence of Nightmare Disorder is unknown.

Course

Nightmares often begin between ages 3 and 6 years. When the frequency is high (e.g., several per week), the dreams may become a source of concern and distress to both children and parents. Most children who develop a nightmare problem outgrow it. In a minority, the dreams may persist at high frequency into adulthood, becoming virtually a lifelong disturbance. Adults with chronic nightmares report similar degrees of subjective sleep disturbance as those who have had nightmares for less than 6 months. A tendency toward amelioration of the disorder in later decades has been described.

Differential Diagnosis

Nightmare Disorder should be differentiated from **Sleep Terror Disorder.** Both disorders include awakenings or partial awakenings with fearfulness and autonomic activation, but can be differentiated by several clinical features. Nightmares typically occur later in the night during REM sleep and produce vivid dream imagery, complete awakenings, mild autonomic arousal, and detailed recall of the event. Sleep terrors typically arise in the first third of the night during stage 3 or 4 NREM sleep and produce either no dream recall or single images without the storylike quality that is typical of nightmares. Sleep terrors lead to partial awakenings in which the individual is confused, disoriented, and only partially responsive and has significant autonomic arousal. In contrast to Nightmare Disorder, the individual with Sleep Terror Disorder has amnesia for the event on awakening in the morning.

Breathing-Related Sleep Disorder can lead to awakenings with autonomic arousal, but these are not accompanied by recall of frightening dreams. Nightmares are a frequent complaint of individuals with **Narcolepsy,** but the presence of excessive sleepiness and cataplexy differentiates this condition from Nightmare Disorder. **Panic Attacks** arising during sleep can also produce abrupt awakenings with autonomic arousal and fearfulness, but the individual does not report frightening dreams and can identify these symptoms as consistent with other Panic Attacks. The presence of complex motor activity during frightening dreams should prompt further evaluation for other Sleep Disorders, such as "REM sleep behavior disorder" (see **Parasomnia Not Otherwise Specified**).

Numerous medications that affect the autonomic nervous system can precipitate nightmares. Examples include L-dopa and other dopaminergic agonists; beta-adrenergic antagonists and other antihypertensive medications; amphetamine, cocaine, and other stimulants; and antidepressant medications. Conversely, withdrawal of medications that suppress REM sleep, such as antidepressant medications and alcohol, can lead to a REM sleep "rebound" accompanied by nightmares. If the nightmares are sufficiently severe to warrant independent clinical attention, a diagnosis of **Substance-Induced Sleep Disorder, Parasomnia Type,** may be considered (see p. 655). Nightmare Disorder also should not be diagnosed if the disturbing dreams arise as a direct physiological effect of a general medical condition (e.g., central nervous system infection, vascular lesions of the brain stem, general medical conditions causing delirium). If the nightmares are sufficiently severe to warrant independent clinical attention, **Sleep Disorder Due to a General Medical Condition, Parasomnia**

Type, may be considered (see p. 651). Although nightmares may frequently occur during a **delirium,** a separate diagnosis of Nightmare Disorder is not given.

Nightmares occur frequently as part of **other mental disorders** (e.g., Posttraumatic Stress Disorder, Schizophrenia, Mood Disorders, other Anxiety Disorders, Adjustment Disorders, and Personality Disorders). If the nightmares occur exclusively during the course of another mental disorder, the diagnosis of Nightmare Disorder is not given.

Many individuals experience an occasional, isolated nightmare. Nightmare Disorder is not diagnosed unless the frequency and severity of nightmares result in significant distress or impairment.

Relationship to the International Classification of Sleep Disorders

Nightmare Disorder corresponds to the diagnosis of Nightmares in the International Classification of Sleep Disorders (ICSD).

Diagnostic criteria for 307.47 Nightmare Disorder

A. Repeated awakenings from the major sleep period or naps with detailed recall of extended and extremely frightening dreams, usually involving threats to survival, security, or self-esteem. The awakenings generally occur during the second half of the sleep period.

B. On awakening from the frightening dreams, the person rapidly becomes oriented and alert (in contrast to the confusion and disorientation seen in Sleep Terror Disorder and some forms of epilepsy).

C. The dream experience, or the sleep disturbance resulting from the awakening, causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The nightmares do not occur exclusively during the course of another mental disorder (e.g., a delirium, Posttraumatic Stress Disorder) and are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

307.46 Sleep Terror Disorder

Diagnostic Features

The essential feature of Sleep Terror Disorder is the repeated occurrence of sleep terrors, that is, abrupt awakenings from sleep usually beginning with a panicky scream or cry (Criterion A). Sleep terrors usually begin during the first third of the major sleep episode and last 1–10 minutes. The episodes are accompanied by autonomic arousal and behavioral manifestations of intense fear (Criterion B). During an episode, the individual is difficult to awaken or comfort (Criterion C). If the individual

awakens after the sleep terror, no dream is recalled, or only fragmentary, single images are recalled. On awakening the following morning, the individual has amnesia for the event (Criterion D). The sleep terror episodes must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). Sleep Terror Disorder should not be diagnosed if the recurrent events are due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (Criterion F). Sleep terrors are also called "night terrors" or pavor nocturnus.

During a typical episode, the individual abruptly sits up in bed screaming or crying, with a frightened expression and autonomic signs of intense anxiety (e.g., tachycardia, rapid breathing, flushing of the skin, sweating, dilation of the pupils, increased muscle tone). The individual is usually unresponsive to the efforts of others to awaken or comfort him or her. If awakened, the person is confused and disoriented for several minutes and recounts a vague sense of terror, usually without dream content. Although fragmentary vivid dream images may occur, a storylike dream sequence (as in nightmares) is not reported. Most commonly, the individual does not awaken fully, but returns to sleep, and has amnesia for the episode on awakening the next morning. Some individuals may vaguely recall having an "episode" during the previous night, but do not have detailed recall. Usually only one episode will occur on any one night, although occasionally several episodes may occur at intervals throughout the night.

For the diagnosis to be made, the individual must experience clinically significant distress or impairment. Embarrassment concerning the episodes can impair social relationships. Individuals may avoid situations in which others might become aware of the disturbance, such as going to camp, visiting friends overnight, or sleeping with bedpartners.

Associated Features and Disorders

Associated descriptive features and mental disorders. The episode is usually accompanied by yelling, screaming, crying, or incoherent vocalizations. The individual may actively resist being held or touched or even demonstrate more elaborate motor activity (e.g., swinging, punching, rising from the bed, or fleeing). These behaviors appear to represent attempts at self-protection or flight from a threat and may result in physical injury. Episodes that simultaneously include features of sleep terror and sleepwalking can occur. Alcohol or sedative use, sleep deprivation, sleep-wake schedule disruptions, fatigue, and physical or emotional stress increase the likelihood of episodes.

Children with Sleep Terror Disorder do not have a higher incidence of psychopathology or mental disorders than does the general population. Psychopathology is more likely to be associated with Sleep Terror Disorder in adults. Sleep Terror Disorder may occur with an increased frequency in individuals with Axis I disorders, particularly Posttraumatic Stress Disorder and Generalized Anxiety Disorder. Personality Disorders may occur in individuals with Sleep Terror Disorder, especially Dependent, Schizoid, and Borderline Personality Disorders. Elevated scores for depression and anxiety have been noted on personality inventories.

Associated laboratory findings. Sleep terrors begin during deep NREM sleep that is characterized by slow-frequency EEG activity (delta). This EEG activity is most prevalent during stages 3 and 4 NREM sleep, which are concentrated in the first third of the major sleep episode. Therefore, sleep terrors are also most likely to occur in the first third of the night. However, episodes can occur during slow-wave sleep at any time, even during daytime naps. The onset of sleep terror episodes is typically heralded by very high voltage EEG delta activity, an increase in muscle tone, and a two-fold to fourfold increase in heart rate, often to over 120 beats per minute. During the episode, the polysomnogram may be obscured with movement artifact. In the absence of such artifact, the EEG typically shows theta or alpha activity during the episode, indicating partial arousal. Individuals with Sleep Terror Disorder may also have abrupt arousals from deep NREM sleep that do not progress to full episodes of sleep terror. Such episodes can include abrupt tachycardia.

Associated physical examination findings and general medical conditions. Fever and sleep deprivation can produce an increased frequency of sleep terror episodes.

Specific Culture, Age, and Gender Features

No reports have provided clear evidence of culturally related differences in the manifestations of Sleep Terror Disorder, although it is likely that the significance and cause attributed to sleep terror episodes will differ between cultures. Older children and adults provide a more detailed recollection of fearful images associated with sleep terrors than do younger children, who are more likely to have complete amnesia or to report only a vague sense of fear. Among children, Sleep Terror Disorder is more common in males than in females. Among adults, the sex ratio is even.

Prevalence

There are limited data on Sleep Terror Disorder in the general population. The prevalence of sleep terror episodes (as opposed to Sleep Terror Disorder in which there is recurrence and distress or impairment) has been estimated at 1%–6% among children and at less than 1% of adults.

Course

Sleep Terror Disorder usually begins in children between ages 4 and 12 years and resolves spontaneously during adolescence. In adults, it most commonly begins between ages 20 and 30 years and often follows a chronic course, with the frequency and severity of episodes waxing and waning over time. The frequency of episodes varies both within and among individuals. Episodes usually occur at intervals of days or weeks but may occur on consecutive nights.

Familial Pattern

Individuals with Sleep Terror Disorder frequently report a positive family history of either sleep terrors or sleepwalking. Some studies indicate a 10-fold increase in the

prevalence of the disorder among first-degree biological relatives. The exact mode of inheritance is unknown.

Differential Diagnosis

Many individuals suffer from isolated episodes of sleep terrors at some time in their lives. The distinction between individual episodes of sleep terrors and Sleep Terror Disorder rests on repeated occurrence, intensity, clinically significant impairment or distress, and the potential for injury to self or others.

Sleep Terror Disorder must be differentiated from other disorders that produce complete or partial awakenings at night or unusual behavior during sleep. The most important differential diagnoses for Sleep Terror Disorder include Nightmare Disorder, Sleepwalking Disorder, other parasomnias (see Parasomnia Not Otherwise Specified), Breathing-Related Sleep Disorder, and seizures occurring during sleep. In contrast to individuals with Sleep Terror Disorder, individuals with **Nightmare Disorder** typically awaken easily and completely, report vivid storylike dreams accompanying the episodes, and tend to have episodes later in the night. The degree of autonomic arousal and motor activity is not as great as that in Sleep Terror Disorder, and recall is more complete. Sleep terrors usually occur during slow-wave sleep, whereas nightmares occur during REM sleep. Parents of children with Sleep Terror Disorder may misinterpret reports of fearfulness and fragmentary imagery reports as nightmares.

Sleepwalking Disorder may be difficult to differentiate from cases of Sleep Terror Disorder that involve prominent motor activity. In fact, the two disorders frequently occur together, and family history commonly involves both disorders. The prototypical case of Sleep Terror Disorder involves a predominance of autonomic arousal and fear, with a lesser degree of motor activity that tends to be abrupt and disorganized. The prototypical case of Sleepwalking Disorder involves little autonomic arousal or fear and a greater degree of organized motor activity.

Parasomnias Not Otherwise Specified include several presentations that can resemble Sleep Terror Disorder. The most common example is "REM sleep behavior disorder," which also produces subjective fear, violent motor activity, and the potential for injury. Because this occurs during REM sleep, it involves vivid storylike dreams, more immediate and complete awakening, and motor activity that clearly follows dream content. "Nocturnal paroxysmal dystonia" also includes awakenings from sleep with motor activity, but this activity is longer in duration, more rhythmic and stereotyped, and not associated with subjective reports or signs of fear.

Hypnagogic hallucinations, experienced sporadically by many otherwise-asymptomatic individuals, as well as more regularly by those with **Narcolepsy,** may be associated with anxiety. Their occurrence at sleep onset, vivid images, and subjective sensation of wakefulness differentiate these episodes from sleep terrors.

Rarely, an individual with a **Breathing-Related Sleep Disorder** may have episodes of awakenings associated with fear and panic that resemble those in Sleep Terror Disorder. The association with snoring, obesity, and respiratory symptoms such as witnessed apneas, an inability to breathe, or choking episodes distinguishes Breathing-Related Sleep Disorder. A single episode of sleep terror can also occur during the slow-wave sleep rebound that follows the abrupt treatment of obstructive

sleep apnea syndrome (e.g., following nasal continuous positive airway pressure [CPAP] therapy).

Seizures that occur during sleep can produce subjective sensations of fear and stereotyped behaviors, followed by confusion and difficulty awakening. Most nocturnal seizures occur at sleep-wake transitions, but they may occur during slow-wave sleep. Incontinence and tonic-clonic movements suggest a seizure disorder, but frontal and temporal lobe seizures can produce more complex behaviors as well. An EEG often reveals interictal findings in individuals with sleep-related seizures, but EEG monitoring during nocturnal sleep may be needed for definitive differential diagnosis. Sleep disruption related to seizures should be diagnosed as **Sleep Disorder Due to a General Medical Condition, Parasomnia Type** (see p. 651). Sleep Disorders Due to a General Medical Condition other than sleep-related seizures may rarely cause unusual behavioral episodes at night. The new onset of abnormal behavior during sleep in a middle-aged or older adult should prompt consideration of a closed head injury or central nervous system pathology such as tumor or infection.

Sleep terror episodes also may be exacerbated or induced by medications such as central nervous system depressants. If episodes are judged to be a direct physiological effect of taking a medication or substance, the disorder should be classified as a **Substance-Induced Sleep Disorder, Parasomnia Type** (see p. 655).

Panic Disorder may also cause abrupt awakenings from deep NREM sleep accompanied by fearfulness, but these episodes produce rapid and complete awakening without the confusion, amnesia, or motor activity typical of Sleep Terror Disorder. Individuals who have Panic Attacks during sleep report that these symptoms are virtually identical to those of Panic Attacks that occur during the day. The presence of Agoraphobia may also help differentiate the two disorders.

Relationship to the International Classification of Sleep Disorders

Sleep Terror Disorder is virtually identical to Sleep Terrors in the International Classification of Sleep Disorders (ICSD). Confusional Arousals, which can occur as an independent disorder or in conjunction with Sleep Terror Disorder, are also described in the ICSD. Confusional Arousals are characterized by brief awakenings from slow-wave sleep with confusion, but without terror or ambulation.

Diagnostic criteria for 307.46 Sleep Terror Disorder

A. Recurrent episodes of abrupt awakening from sleep, usually occurring during the first third of the major sleep episode and beginning with a panicky scream.

B. Intense fear and signs of autonomic arousal, such as tachycardia, rapid breathing, and sweating, during each episode.

C. Relative unresponsiveness to efforts of others to comfort the person during the episode.

D. No detailed dream is recalled and there is amnesia for the episode.

E. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

307.46 Sleepwalking Disorder

Diagnostic Features

The essential feature of Sleepwalking Disorder is repeated episodes of complex motor behavior initiated during sleep, including rising from bed and walking about. Sleepwalking episodes begin during slow-wave sleep and therefore most often occur during the first third of the night (Criterion A). During episodes, the individual has reduced alertness and responsiveness, a blank stare, and relative unresponsiveness to communication with others or efforts to be awakened by others (Criterion B). If awakened during the episode (or on awakening the following morning), the individual has limited recall for the events of the episode (Criterion C). After the episode, there may initially be a brief period of confusion or difficulty orienting, followed by full recovery of cognitive function and appropriate behavior (Criterion D). The sleepwalking must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). Sleepwalking Disorder should not be diagnosed if the behavior is due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (Criterion F).

Sleepwalking episodes can include a variety of behaviors. In mild episodes (sometimes called "confusional arousals"), the individual may simply sit up in bed, look about, or pick at the blanket or sheet. More typically, the individual actually gets out of bed and may walk into closets, out of the room, up and down stairs, and even out of buildings. Individuals may use the bathroom, eat, and talk during episodes. Running and frantic attempts to escape some apparent threat can also occur. Most behaviors during sleepwalking episodes are routine and of low complexity. However, cases of unlocking doors and even operating machinery have been reported. Particularly in

childhood, sleepwalking can also include inappropriate behavior (e.g., urinating in a closet). Most episodes last for several minutes to a half hour.

Sleepwalking episodes can terminate in spontaneous arousals followed by a brief period of confusion, or the individual may return to bed and continue to sleep until the morning. Not uncommonly, the individual may awaken the next morning in another place, or with evidence of having performed some activity during the night, but with complete amnesia for the event. Some episodes may be followed by vague recall of fragmentary dream images, but usually not by typical storylike dreams.

During sleepwalking episodes, individuals may talk or even respond to others' questions. However, their articulation is poor, and true dialogue is rare. Individuals may respond to others' requests to cease their activity and return to bed. However, these behaviors are performed with reduced levels of alertness, and awakening an individual from a sleepwalking episode is typically very difficult. If awakened, the individual remains confused for several minutes and then returns to a normal state of alertness.

For the diagnosis to be made, the individual must experience clinically significant distress or impairment. Individuals may avoid situations that would reveal their behavior to others (e.g., children may avoid visiting friends or going to summer camp; adults may avoid sleeping with bedpartners, going on vacation, or staying away from home). Social isolation or occupational difficulties can result.

Associated Features and Disorders

Associated descriptive features and mental disorders. Internal stimuli (e.g., a distended bladder) or external stimuli (e.g., noises) can increase the likelihood of a sleepwalking episode, as can psychosocial stressors and alcohol or sedative use. Some individuals with sleepwalking also report episodes of eating during the night, most often with complete or partial amnesia. They may find evidence of their eating only the next morning. Individuals can injure themselves during sleepwalking episodes by bumping into objects, walking on stairs, going outside, and even walking out of windows. The risk of injury further increases if sleepwalking episodes also include features of sleep terrors, with an attendant fleeing or striking out. Individuals with Sleepwalking Disorder and Sleep Terror Disorder can also injure others during episodes.

Other parasomnias associated with non–rapid eye movement (NREM) sleep (e.g., Sleep Terror Disorder) can also occur in individuals with Sleepwalking Disorder. Sleepwalking Disorder in children usually is not associated with other mental disorders, but in adults it may be associated with Personality Disorders, Mood Disorders, or Anxiety Disorders.

Associated laboratory findings. Polysomnography, using routine procedures with the addition of audiovisual monitoring, can document episodes of sleepwalking. The majority of sleepwalking episodes begin within the first few hours of sleep, typically during NREM stage 3 or 4 sleep, although some individuals (e.g., older adults) may have episodes during NREM stage 2 sleep. Preceding the episode, the EEG often shows rhythmic ("hypersynchronous"), high-voltage delta activity that persists during the arousal. EEG signs of arousal, such as alpha activity, may also ap-

pear at the beginning of the episode. Most commonly, the EEG is obscured by movement artifact during the actual episode. Heart rate and respiratory rate may increase at the beginning of the episode. These findings may occur with a full sleepwalking episode or with a more minor behavioral event (such as a confusional arousal). Other polysomnographic findings may include an increased number of transitions out of stages 3 and 4 sleep and reduced sleep efficiency. Other polysomnographic findings may include an increased number of transitions out of stages 3 and 4 sleep, increased awakenings during NREM sleep, and reduced sleep efficiency. Sleep-stage architecture may show an increase in NREM stages 3 and 4 sleep but is otherwise unremarkable. Sleep apnea and periodic limb movements are seen in a minority of individuals with Sleepwalking Disorder.

Associated physical examination findings and general medical conditions. Fever or sleep deprivation can increase the frequency of sleepwalking episodes. Obstructive sleep apnea syndrome, periodic limb movement disorder, and other disorders that produce severe disruption of slow-wave sleep can also be associated with sleepwalking episodes. An association has been noted between Sleepwalking Disorder and migraine headaches, Narcolepsy, and other neurological conditions in a subset of individuals.

Specific Culture, Age, and Gender Features

No reports have provided clear evidence of culturally related differences in the manifestations of Sleepwalking Disorder, but it is likely that the significance and causes attributed to sleepwalking differ among cultures. In clinical samples, violent activity during sleepwalking episodes is more likely to occur in adults (particularly in men), whereas eating during sleepwalking episodes is more commonly seen in women. Sleepwalking Disorder occurs more often in females during childhood but more often in males during adulthood.

Prevalence

Between 10% and 30% of children have had at least one episode of sleepwalking, and 2%–3% sleepwalk often. The prevalence of Sleepwalking Disorder (marked by repeated episodes and impairment or distress) is much lower, probably in the range of 1%–5%. Epidemiological surveys report the prevalence of sleepwalking episodes (not Sleepwalking Disorder) to be 1.0%–7.0% among adults, with weekly to monthly episodes occurring in 0.5%–0.7%.

Course

Sleepwalking can occur at any time after a child is able to walk, but episodes most commonly occur for the first time between ages 4 and 8 years. The peak prevalence occurs at about age 12. Episodes rarely occur for the first time in adults, although some associated behaviors such as nocturnal eating may begin several years after the sleepwalking itself. The onset of Sleepwalking Disorder in adults with no history of sleepwalking as children should prompt a search for specific etiologies such as sub-

stance use or a neurological condition. The majority of adults with Sleepwalking Disorder have a history of episodes during childhood as well. Sleepwalking in childhood usually disappears spontaneously during early adolescence, typically by age 15 years. Less commonly, episodes may have a recurrent course, with return of episodes in early adulthood after cessation of episodes in late childhood. Sleepwalking Disorder in adults most often follows a chronic, waxing and waning course. Sleepwalking episodes may occur as isolated events in individuals of any age, but the most common pattern is repeated episodes occurring over a period of several years.

Familial Pattern

Sleepwalking Disorder aggregates among family members. A family history for sleepwalking or sleep terrors has been reported in up to 80% of individuals who sleepwalk. The risk for sleepwalking is further increased (to as much as 60% of offspring) when both parents have a history of the disorder. Genetic transmission is suggested by an increased prevalence of monozygotic, as opposed to dizygotic, twins, but the exact mode of inheritance is not known.

Differential Diagnosis

Many children have isolated or infrequent episodes of sleepwalking, either with or without precipitating events. The exact boundary between **nonclinically significant sleepwalking episodes** and Sleepwalking Disorder is indistinct. Frequent episodes, injuries, more active or violent behavior, and social impairment resulting from sleepwalking are likely to lead the child's parents to seek help and warrant a diagnosis of Sleepwalking Disorder. Episodes that have persisted from childhood to late adolescence, or that occur de novo in adults, are more likely to warrant a diagnosis of Sleepwalking Disorder.

It can be difficult clinically to distinguish Sleepwalking Disorder from **Sleep Terror Disorder** when there is an attempt to "escape" from the terrifying stimulus. In both cases, the individual shows movement, difficulty awakening, and amnesia for the event. An initial scream, signs of intense fear and panic, and autonomic arousal are more characteristic of Sleep Terror Disorder. Sleepwalking Disorder and Sleep Terror Disorder may occur in the same individual, and in such cases both should be diagnosed.

Breathing-Related Sleep Disorder, especially the obstructive sleep apnea syndrome, can also produce confusional arousals with subsequent amnesia. However, Breathing-Related Sleep Disorder is also characterized by characteristic symptoms of snoring, breathing pauses, and daytime sleepiness. In some individuals, Breathing-Related Sleep Disorder may precipitate episodes of sleepwalking.

"REM sleep behavior disorder" is another Parasomnia (see **Parasomnia Not Otherwise Specified**) that may be difficult to distinguish from Sleepwalking Disorder. REM sleep behavior disorder is characterized by episodes of prominent, complex movements, often involving personal injury. In contrast to Sleepwalking Disorder, REM sleep behavior disorder occurs during rapid eye movement (REM) sleep, often in the later part of the night. Individuals with REM sleep behavior disorder awaken easily and report more detailed and vivid dream content than do individuals with

Sleepwalking Disorder. A small number of individuals may have confusional arousals with motor activity that occur during both NREM and REM sleep. The definitive diagnosis in such cases should be based on a careful evaluation of clinical, polysomnographic, and other laboratory findings.

A variety of other behaviors can occur with partial arousals from sleep. Confusional arousals resemble sleepwalking episodes in all respects except the actual movement out of the bed. "Sleep drunkenness" is a state in which the individual shows a prolonged transition from sleep to wakefulness in the morning. It may be difficult to arouse the individual, who may violently resist efforts to awaken him or her. Again, ambulation or other more complex behaviors distinguish Sleepwalking Disorder. However, both confusional arousals and sleep drunkenness may occur in individuals with Sleepwalking Disorder.

Sleep-related seizures can produce episodes of unusual behavior that occur only during sleep. The individual is unresponsive and is amnestic for the episode. Typically, sleep-related epilepsy produces more stereotypical, perseverative, low-complexity movements than those in sleepwalking. In most cases, individuals with sleep-related epilepsy also have similar episodes during wakefulness. The EEG shows features of epilepsy, including paroxysmal activity during the episodes and interictal features at other times. However, the presence of sleep-related seizures does not preclude the presence of sleepwalking episodes. Sleep-related epilepsy should be diagnosed as **Sleep Disorder Due to General Medical Condition, Parasomnia Type** (see p. 651).

Sleepwalking can be induced by use of, or withdrawal from, substances or medications (e.g., alcohol, benzodiazepines, opiates, cocaine, nicotine, antipsychotics, tricyclic antidepressants, chloral hydrate). In such cases, **Substance-Induced Sleep Disorder, Parasomnia Type,** should be diagnosed (see p. 655).

Dissociative Fugue bears superficial similarities to Sleepwalking Disorder. Fugue is rare in children, typically begins when the individual is awake, lasts hours or days, and is not characterized by disturbances of consciousness. Sleepwalking must also be distinguished from **Malingering** or **other voluntary behavior occurring during wakefulness,** although in some cases such distinctions may be difficult. Features that suggest Sleepwalking Disorder include a positive childhood history, low-complexity or stereotyped behavior during sleepwalking episodes, the absence of secondary gain to the individual from his or her nocturnal behavior, and the presence of typical polysomnographic findings such as repeated arousals from NREM sleep. Furthermore, it may be difficult for the individual to convincingly counterfeit the appearance or behavior of sleepwalking under direct observation or in a video recording made in the sleep laboratory.

Relationship to the International Classification of Sleep Disorders

Sleepwalking Disorder is virtually identical to Sleepwalking as described in the International Classification of Sleep Disorders (ICSD). The ICSD includes two other disorders that may have features similar to sleepwalking: Confusional Arousals and Nocturnal Eating (Drinking) Syndrome.

Diagnostic criteria for 307.46 Sleepwalking Disorder

A. Repeated episodes of rising from bed during sleep and walking about, usually occurring during the first third of the major sleep episode.

B. While sleepwalking, the person has a blank, staring face, is relatively unresponsive to the efforts of others to communicate with him or her, and can be awakened only with great difficulty.

C. On awakening (either from the sleepwalking episode or the next morning), the person has amnesia for the episode.

D. Within several minutes after awakening from the sleepwalking episode, there is no impairment of mental activity or behavior (although there may initially be a short period of confusion or disorientation).

E. The sleepwalking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

307.47 Parasomnia Not Otherwise Specified

The Parasomnia Not Otherwise Specified category is for disturbances that are characterized by abnormal behavioral or physiological events during sleep or sleep-wake transitions, but that do not meet criteria for a more specific Parasomnia. Examples include

1. REM sleep behavior disorder: motor activity, often of a violent nature, that arises during rapid eye movement (REM) sleep. Unlike sleepwalking, these episodes tend to occur later in the night and are associated with vivid dream recall.
2. Sleep paralysis: an inability to perform voluntary movement during the transition between wakefulness and sleep. The episodes may occur at sleep onset (hypnagogic) or with awakening (hypnopompic). The episodes are usually associated with extreme anxiety and, in some cases, fear of impending death. Sleep paralysis occurs commonly as an ancillary symptom of Narcolepsy and, in such cases, should not be coded separately.
3. Situations in which the clinician has concluded that a Parasomnia is present but is unable to determine whether it is primary, due to a general medical condition, or substance induced.

Sleep Disorders Related to Another Mental Disorder

307.42 Insomnia Related to Another Mental Disorder
307.44 Hypersomnia Related to Another Mental Disorder

Diagnostic Features

The essential feature of Insomnia Related to Another Mental Disorder and Hypersomnia Related to Another Mental Disorder is the presence of either insomnia or hypersomnia that is judged to be related temporally and causally to another mental disorder. Insomnia or Hypersomnia that is the direct physiological consequence of a substance is not included here. Such presentations would be diagnosed as Substance-Induced Sleep Disorder (see p. 655). Insomnia Related to Another Mental Disorder is characterized by a complaint of difficulty falling asleep, frequent awakenings during the night, or a marked feeling of nonrestorative sleep that has lasted for at least 1 month and is associated with daytime fatigue or impaired daytime functioning (Criterion A). Hypersomnia Related to Another Mental Disorder is characterized by a complaint of either prolonged nighttime sleep or repeated daytime sleep episodes for at least 1 month (Criterion A). In both Insomnia and Hypersomnia Related to Another Mental Disorder, the sleep symptoms cause significant distress or impairment in social, occupational, or other important areas of functioning (Criterion B). The insomnia or hypersomnia is not better accounted for by another Sleep Disorder (e.g., Narcolepsy, Breathing-Related Sleep Disorder, or a Parasomnia) and hypersomnia is not better accounted for by an inadequate amount of sleep (Criterion D). The sleep disturbance must not be due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (Criterion E).

Sleep disturbances are common features of other mental disorders. An additional diagnosis of Insomnia or Hypersomnia Related to Another Mental Disorder is made only when the sleep disturbance is a predominant complaint and is sufficiently severe to warrant independent clinical attention (Criterion C). Individuals with this type of insomnia or hypersomnia usually focus on their sleep disturbance to the exclusion of the symptoms characteristic of the related mental disorder, whose presence may become apparent only after specific and persistent questioning. Not infrequently, they attribute their symptoms of mental disorder to the fact that they have slept poorly.

Many mental disorders may at times involve insomnia or hypersomnia as the predominant problem. Individuals in a Major Depressive Episode or who have Dysthymic Disorder often complain of difficulty falling asleep or staying asleep or early morning awakening with inability to return to sleep. Hypersomnia Related to Mood Disorder is more often associated with Bipolar Mood Disorder, Most Recent Episode

Depressed, or a Major Depressive Episode, With Atypical Features. Individuals with Generalized Anxiety Disorder often report difficulty falling asleep and may awaken with anxious ruminations in the middle of the night. Some individuals with Panic Disorder have nocturnal Panic Attacks that can lead to insomnia. Significant insomnia is often seen during exacerbations of Schizophrenia and other Psychotic Disorders but is rarely the predominant complaint. Other mental disorders that may be related to insomnia include Adjustment Disorders, Somatoform Disorders, and Personality Disorders.

Recording Procedures

The name of the diagnosis begins with the type of sleep disturbance (i.e., insomnia or hypersomnia) followed by the name of the specific Axis I or Axis II disorder that it is related to (e.g., 307.42 Insomnia Related to Major Depressive Disorder) on Axis I. The specific related mental disorder should also be coded on Axis I or Axis II as appropriate.

Associated Features and Disorders

Associated descriptive features and mental disorders. Because, by definition, the criteria are met for the related mental disorder, the associated features of Insomnia or Hypersomnia Related to Another Mental Disorder include the characteristic and associated features of the related mental disorder.

Individuals with Insomnia Related to Another Mental Disorder may demonstrate the same type of conditioned arousal and negative conditioning that individuals with Primary Insomnia demonstrate. For instance, they will note increased anxiety as bedtime approaches, improved sleep when taken out of the usual sleep environment, and a tendency to spend too much time in bed. They may also have a history of multiple or inappropriate medication treatments for their insomnia complaints. Individuals with Hypersomnia Related to Another Mental Disorder will frequently emphasize symptoms of fatigue, "leaden paralysis," or complete lack of energy. On careful questioning, these individuals may be more distressed by such fatigue-related symptoms than by true sleepiness. They may also have a history of inappropriate use of stimulant medications, including caffeine.

Associated laboratory findings. Characteristic (but not diagnostic) polysomnographic findings in Major Depressive Episode include 1) sleep continuity disturbance, such as prolonged sleep latency, increased intermittent wakefulness, and early morning awakening; 2) reduced non–rapid eye movement (NREM) stages 3 and 4 sleep (slow-wave sleep), with a shift in slow-wave activity away from the first NREM period; 3) decreased rapid eye movement (REM) latency (i.e., shorter duration of the first NREM period); 4) increased REM density (i.e., the number of actual eye movements during REM); and 5) increased duration of REM sleep early in the night. Sleep abnormalities may be evident in 40%–60% of outpatients and in up to 90% of inpatients with a Major Depressive Episode. Evidence suggests that most of these abnormalities persist after clinical remission and may precede the onset of the initial Major Depressive Episode.

Polysomnographic findings in Manic Episodes are similar to those found in Major Depressive Episodes. In Schizophrenia, REM sleep is diminished early in the course of an acute exacerbation, with a gradual return toward normal values as clinical status improves. REM latency may be reduced. Total sleep time is often severely diminished in Schizophrenia, and slow-wave sleep is typically reduced during exacerbations. Individuals with Panic Disorder may have paroxysmal awakenings on entering stages 3 and 4 NREM sleep; these awakenings are accompanied by tachycardia, increased respiratory rate, and cognitive and emotional symptoms with Panic Attacks. Most other mental disorders produce nonspecific patterns of sleep disturbance (e.g., prolonged sleep latency or frequent awakenings).

Laboratory testing of daytime sleepiness by the Multiple Sleep Latency Test in individuals with Hypersomnia Related to Another Mental Disorder often shows normal or only mild levels of physiological sleepiness compared with individuals with Primary Hypersomnia or Narcolepsy.

Associated physical examination findings and general medical conditions. Individuals with Insomnia or Hypersomnia Related to Another Mental Disorder may appear tired, fatigued, or haggard during routine examination. The general medical conditions associated with these Sleep Disorders are the same as those associated with the underlying mental disorder.

Specific Culture, Age, and Gender Features

In some cultures, sleep complaints may be viewed as relatively less stigmatizing than mental disorders. Therefore, individuals from some cultural backgrounds may be more likely to present with complaints of insomnia or hypersomnia rather than with other symptoms (e.g., depression, anxiety).

Children and adolescents with Major Depressive Disorder generally present with less subjective sleep disturbance and fewer polysomnographic changes than do older adults. In general, hypersomnia is a more common feature of Depressive Disorders in adolescents and young adults and insomnia is more common in older adults.

Sleep Disorders Related to Another Mental Disorder are more prevalent in females than in males. This difference probably relates to the increased prevalence of Mood and Anxiety Disorders in women rather than to any particular difference in susceptibility to sleep problems.

Prevalence

Sleep problems are extremely common in all types of mental disorders, but there are no accurate estimates of the percentage of individuals who present primarily because of sleep disruption. Insomnia Related to Another Mental Disorder is the most frequent diagnosis (35%–50%) among individuals presenting to sleep disorders centers for evaluation of chronic insomnia. Hypersomnia Related to Another Mental Disorder is a much less frequent diagnosis (fewer than 5%) among individuals evaluated for hypersomnia at sleep disorders centers.

Course

The course of Sleep Disorders Related to Another Mental Disorder generally follows the course of the underlying mental disorder itself. The sleep disturbance may be one of the earliest symptoms to appear in individuals who subsequently develop an associated mental disorder. Symptoms of insomnia or hypersomnia often fluctuate considerably over time. For many individuals with depression, particularly those treated with medications, sleep disturbance may improve rapidly, often more quickly than other symptoms of the underlying mental disorder. On the other hand, other individuals have persistent or intermittent insomnia even after the other symptoms of their Major Depressive Disorder remit. Individuals with Bipolar Disorder often have distinctive sleep-related symptoms depending on the nature of the current episode. During Manic Episodes, individuals experience hyposomnia, although they rarely complain about their inability to sleep. On the other hand, such individuals may have marked distress about hypersomnia during Major Depressive Episodes. Individuals with Psychotic Disorders most often have a notable worsening in sleep early during the course of an acute exacerbation, but then report improvement as psychotic symptoms abate.

Differential Diagnosis

Insomnia or Hypersomnia Related to Another Mental Disorder should not be diagnosed in every individual with a mental disorder who also has sleep-related symptoms. A diagnosis of Insomnia or Hypersomnia Related to Another Mental Disorder should be made only when sleep symptoms are severe and are an independent focus of clinical attention. No independent sleep disorder diagnosis is warranted for most individuals with **Major Depressive Disorder** who report difficulties falling or staying asleep in the middle of the night. However, if the individual primarily complains of sleep disturbance or if the insomnia is out of proportion to other symptoms, then an additional diagnosis of Insomnia Related to Another Mental Disorder may be warranted.

Distinguishing **Primary Insomnia** or **Primary Hypersomnia** from Insomnia or Hypersomnia Related to Another Mental Disorder can be especially difficult in individuals who present with both clinically significant sleep disturbance and other symptoms of a mental disorder. The diagnosis of Insomnia or Hypersomnia Related to Another Mental Disorder is based on three judgments. First, the insomnia or hypersomnia must be judged to be attributable to the mental disorder (e.g., the insomnia or hypersomnia occurs exclusively during the mental disorder). Second, the insomnia or hypersomnia must be the predominant complaint and must be sufficiently severe to warrant independent clinical attention. Third, the symptom presentation should meet the full criteria for another mental disorder. A diagnosis of Primary Insomnia or Primary Hypersomnia is appropriate when (as is often the case) the insomnia or hypersomnia is accompanied by symptoms (e.g., anxiety, depressed mood) that do not meet criteria for a specific mental disorder. A diagnosis of Primary Insomnia is also appropriate for individuals with chronic insomnia who later develop a Mood or Anxiety Disorder. If symptoms of insomnia or hypersomnia persist long after the other symptoms of the related mental disorder have remitted completely,

the diagnosis would be changed from Insomnia or Hypersomnia Related to Another Mental Disorder to Primary Insomnia or Primary Hypersomnia.

Insomnia or Hypersomnia Related to Another Mental Disorder is not diagnosed if the presentation is better accounted for by **another Sleep Disorder** (e.g., Narcolepsy, Breathing-Related Sleep Disorder, or a Parasomnia).

Insomnia or Hypersomnia Related to Another Mental Disorder must be distinguished from a **Sleep Disorder Due to a General Medical Condition.** The diagnosis is Sleep Disorder Due to a General Medical Condition when the sleep disturbance is judged to be a direct physiological consequence of a specific general medical condition (e.g., pheochromocytoma, hyperthyroidism). This determination is based on history, laboratory findings, and physical examination (see p. 651 for further discussion). A **Substance-Induced Sleep Disorder** is distinguished from Insomnia or Hypersomnia Related to Another Mental Disorder by the fact that a substance (i.e., a drug of abuse, a medication) is judged to be etiologically related to the sleep disturbance (see p. 655 for further discussion). For example, insomnia that occurs only in the context of heavy coffee consumption would be diagnosed as Caffeine-Induced Sleep Disorder, Insomnia Type.

Sleep Disorders Related to Another Mental Disorder must be differentiated from **normal sleep patterns,** as well as from other Sleep Disorders. Although complaints of occasional insomnia or hypersomnia are common in the general population, they are not usually accompanied by the other signs and symptoms of a mental disorder. Transient sleep disturbances are common reactions to stressful life events and generally do not warrant a diagnosis. A separate diagnosis of Insomnia or Hypersomnia Related to Adjustment Disorder should be considered only when the sleep disturbance is particularly severe and prolonged.

Relationship to the International Classification of Sleep Disorders

The International Classification of Sleep Disorders (ICSD) includes analogous diagnoses for Sleep Disorders Related to Another Mental Disorder and specifically lists Psychoses, Mood Disorders, Anxiety Disorders, Panic Disorder, and Alcoholism.

Diagnostic criteria for 307.42 Insomnia Related to . . . *[Indicate the Axis I or Axis II disorder]*

A. The predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month that is associated with daytime fatigue or impaired daytime functioning.

B. The sleep disturbance (or daytime sequelae) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The insomnia is judged to be related to another Axis I or Axis II disorder (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, Adjustment Disorder With Anxiety) but is sufficiently severe to warrant independent clinical attention.

D. The disturbance is not better accounted for by another Sleep Disorder (e.g., Narcolepsy, Breathing-Related Sleep Disorder, a Parasomnia).

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Diagnostic criteria for 307.44 Hypersomnia Related to . . . *[Indicate the Axis I or Axis II disorder]*

A. The predominant complaint is excessive sleepiness for at least 1 month as evidenced by either prolonged sleep episodes or daytime sleep episodes that occur almost daily.

B. The excessive sleepiness causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The hypersomnia is judged to be related to another Axis I or Axis II disorder (e.g., Major Depressive Disorder, Dysthymic Disorder) but is sufficiently severe to warrant independent clinical attention.

D. The disturbance is not better accounted for by another Sleep Disorder (e.g., Narcolepsy, Breathing-Related Sleep Disorder, a Parasomnia) or by an inadequate amount of sleep.

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Other Sleep Disorders

780.xx Sleep Disorder Due to a General Medical Condition

Diagnostic Features

The essential feature of Sleep Disorder Due to a General Medical Condition is a prominent disturbance in sleep that is severe enough to warrant independent clinical attention (Criterion A) and is due to a general medical condition. Symptoms may include insomnia, hypersomnia, a Parasomnia, or some combination of these. There must be evidence from the history, physical examination, or laboratory findings that the sleep disturbance is the direct physiological consequence of a general medical condition (Criterion B). The disturbance is not better accounted for by another mental disorder, such as Adjustment Disorder, in which the stressor is a serious general medical condition (Criterion C). The diagnosis is not made if the sleep disturbance occurs only during the course of a delirium (Criterion D). By convention, sleep disturbances due to a Sleep-Related Breathing Disorder (e.g., sleep apnea) or to Narcolepsy are not included in this category (Criterion E). The sleep symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion F).

In determining whether the sleep disturbance is due to a general medical condition, the clinician must first establish the presence of a general medical condition. Further, the clinician must establish that the sleep disturbance is etiologically related to the general medical condition through a physiological mechanism. A careful and comprehensive assessment of multiple factors is necessary to make this judgment. Although there are no infallible guidelines for determining whether the relationship between the sleep disturbance and the general medical condition is etiological, several considerations provide some guidance in this area. One consideration is the presence of a temporal association between the onset, exacerbation, or remission of the general medical condition and that of the sleep disturbance. A second consideration is the presence of features that are atypical of primary Sleep Disorders (e.g., atypical age at onset or course or absence of family history). Evidence from the literature that suggests that there can be a direct association between the general medical condition in question and the development of a sleep disturbance can provide a useful context in the assessment of a particular situation. In addition, the clinician must also judge that the disturbance is not better accounted for by a primary Sleep Disorder, a Substance-Induced Sleep Disorder, or other primary mental disorders (e.g., Adjustment Disorder). This determination is explained in greater detail in the "Mental Disorders Due to a General Medical Condition" section (p. 181).

Subtypes

The subtypes listed below can be used to indicate which of the following symptom presentations predominates. The clinical presentation of the specific Sleep Disorder

Due to a General Medical Condition may resemble that of the analogous primary Sleep Disorder. However, the full criteria for the analogous primary Sleep Disorder do not need to be met to assign a diagnosis of Sleep Disorder Due to a General Medical Condition.

Insomnia Type. This subtype refers to a sleep complaint characterized primarily by difficulty falling asleep, difficulty maintaining sleep, or a feeling of nonrestorative sleep.

Hypersomnia Type. This subtype is used when the predominant complaint is one of excessively long nocturnal sleep or of excessive sleepiness during waking hours.

Parasomnia Type. This subtype refers to a sleep disturbance characterized primarily by abnormal behavioral events that occur in association with sleep or sleep transitions.

Mixed Type. This subtype should be used to designate a sleep problem due to a general medical condition characterized by multiple sleep symptoms but no symptom clearly predominates.

Recording Procedures

In recording the diagnosis of Sleep Disorder Due to a General Medical Condition, the clinician should note both the specific phenomenology of the disturbance, including the appropriate subtype, and the specific general medical condition judged to be causing the disturbance on Axis I (e.g., 780.52 Sleep Disorder Due to Thyrotoxicosis, Insomnia Type). The ICD-9-CM code for the general medical condition should also be noted on Axis III (e.g., 242.9 thyrotoxicosis). (See Appendix G for a list of selected ICD-9-CM diagnostic codes for general medical conditions.)

Associated Features and Disorders

Associated laboratory findings. Laboratory findings are consistent with the underlying general medical condition. There are no polysomnographic findings that are specific to the entire group of Sleep Disorders Due to a General Medical Condition. Most general medical conditions cause a decrease in total sleep duration, an increase in awakenings, a decrease in slow-wave sleep, and (less consistently) a decrease in rapid eye movement (REM) sleep or phasic REM density. Some medical conditions produce more specific polysomnographic findings. For example, individuals with fibromyalgia syndrome complain of nonrestorative sleep and often have a distinct pattern of alpha EEG activity during non–rapid eye movement (NREM) sleep. Sleep-related seizures result in specific EEG discharges that are consistent with the underlying seizure type.

Associated physical examination findings and general medical conditions. Individuals with a Sleep Disorder Due to a General Medical Condition are expected to have the typical physical findings of the underlying general medical condition. Sleep disturbances may result from a variety of general medical and neurological conditions including (but not limited to) degenerative neurological illnesses (e.g., Parkinson's disease, Huntington's disease), cerebrovascular disease (e.g., insomnia

following vascular lesions to the upper brain stem), endocrine conditions (e.g., hypo- or hyperthyroidism, hypo- or hyperadrenocorticism), viral and bacterial infections (e.g., hypersomnia related to viral encephalitis), coughing related to pulmonary disease other than sleep-related breathing conditions (e.g., chronic bronchitis), and pain from musculoskeletal disease (e.g., rheumatoid arthritis, fibromyalgia). General medical conditions in which hypersomnia may present as a core feature of the illness include myotonic dystrophy and Prader-Willi syndrome.

Differential Diagnosis

Sleep disturbances are extremely common in the context of a **delirium**; therefore, a separate diagnosis of Sleep Disorder Due to a General Medical Condition is not given if the disturbance occurs exclusively during the course of the delirium. In contrast, a diagnosis of Sleep Disorder Due to a General Medical Condition may be given in addition to a diagnosis of **dementia** if the sleep disturbance is a direct etiological consequence of the pathological process causing the dementia and the sleep disturbance is a prominent part of the clinical presentation.

Sleep Disorder Due to a General Medical Condition must be differentiated from expected disruptions in sleep patterns, primary Sleep Disorders, Sleep Disorders Related to Another Mental Disorder, and Substance-Induced Sleep Disorders. Many individuals experience **sleep disruption during the course of a general medical or neurological condition.** In the majority of cases, such complaints do not merit an additional diagnosis of a Sleep Disorder. Rather, a diagnosis of Sleep Disorder Due to a General Medical Condition should be reserved for cases in which the sleep disturbance is a very prominent clinical feature, atypical symptoms are present, or the individual is sufficiently distressed by the sleep symptom or attendant impairment that specific treatment for this disturbance is required.

Sleep Disorders Due to a General Medical Condition are characterized by symptoms similar to those in **primary Sleep Disorders.** The differential diagnosis rests not on specific symptoms but rather on the presence or absence of a medical condition judged to be etiologically related to the sleep complaint. In the specific cases of **Narcolepsy** and **Breathing-Related Sleep Disorder,** the underlying etiology of the sleep disturbance is assumed to be a general medical condition. However, in these two specific examples, the general medical condition does not exist independent of sleep symptoms. For this reason, these two disorders are included in the "Primary Sleep Disorders" section.

Differentiating a Sleep Disorder Due to a General Medical Condition from **Substance-Induced Sleep Disorder** can prove very difficult. In many cases, individuals with a significant general medical condition often take medication for that condition; these medications in turn may cause sleep-related symptoms. For example, an individual may have sleep disruption related to asthma. However, that individual may also be treated with theophylline preparations, which in some cases can themselves cause sleep disturbance. Differentiating a Sleep Disorder Due to a General Medical Condition from a Substance-Induced Sleep Disorder often rests on chronology, response to treatment or discontinuation of medications, and longitudinal course. In some cases, concurrent diagnoses of Sleep Disorder Due to a General Medical Condition and Substance-Induced Sleep Disorder may be appropriate. In cases in which a drug of abuse is suspected to be the cause for the Sleep Disorder, a urine or

blood drug screen may help to differentiate this problem from a Sleep Disorder Due to a General Medical Condition.

If the clinician cannot determine whether the sleep disturbance is primary, related to another mental disorder, due to a general medical condition, or substance induced, the appropriate diagnosis is Dyssomnia or Parasomnia Not Otherwise Specified.

Relationship to the International Classification of Sleep Disorders

The International Classification of Sleep Disorders (ICSD) contains the general section "Medical/Psychiatric Sleep Disorders." Specific diagnoses are presented for Sleep Disorders that are associated with neurological disorders (with 7 examples listed) and Sleep Disorders that are associated with other medical disorders (with 7 examples listed). Although only 14 medical/neurological disorders are specifically cited in the ICSD, the clinician may diagnose a Sleep Disorder associated with any other medical disorder simply by using the appropriate ICD-9-CM codes.

Diagnostic criteria for 780.xx Sleep Disorder Due to . . . *[Indicate the General Medical Condition]*

A. A prominent disturbance in sleep that is sufficiently severe to warrant independent clinical attention.

B. There is evidence from the history, physical examination, or laboratory findings that the sleep disturbance is the direct physiological consequence of a general medical condition.

C. The disturbance is not better accounted for by another mental disorder (e.g., an Adjustment Disorder in which the stressor is a serious medical illness).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance does not meet the criteria for Breathing-Related Sleep Disorder or Narcolepsy.

F. The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify type:

.52 Insomnia Type: if the predominant sleep disturbance is insomnia

.54 Hypersomnia Type: if the predominant sleep disturbance is hypersomnia

.59 Parasomnia Type: if the predominant sleep disturbance is a Parasomnia

.59 Mixed Type: if more than one sleep disturbance is present and none predominates

Coding note: Include the name of the general medical condition on Axis I, e.g., 780.52 Sleep Disorder Due to Chronic Obstructive Pulmonary Disease, Insomnia Type; also code the general medical condition on Axis III (see Appendix G for codes).

Substance-Induced Sleep Disorder

Diagnostic Features

The essential feature of Substance-Induced Sleep Disorder is a prominent disturbance in sleep that is sufficiently severe to warrant independent clinical attention (Criterion A) and is judged to be due to the direct physiological effects of a substance (i.e., a drug of abuse, a medication, or toxin exposure) (Criterion B). Depending on the substance involved, one of four types of sleep disturbance may be noted. Insomnia and Hypersomnia Types are most common, and Parasomnia Type is seen less often. A Mixed Type may also be noted when more than one type of sleep disturbance is present and none predominates. The disturbance must not be better accounted for by a mental disorder (e.g., another Sleep Disorder) that is not substance induced (Criterion C). The diagnosis is not made if the sleep disturbance occurs only during the course of a delirium (Criterion D). The symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). This diagnosis should be made instead of a diagnosis of Substance Intoxication or Substance Withdrawal only when the symptoms are in excess of those usually associated with the intoxication or withdrawal syndrome and when the symptoms are sufficiently severe to warrant independent clinical attention. For a more detailed discussion of Substance-Related Disorders, see p. 191.

A Substance-Induced Sleep Disorder is distinguished from a primary Sleep Disorder and from Insomnia or Hypersomnia Related to Another Mental Disorder by considering the onset and course. For drugs of abuse, there must be evidence from the history, physical examination, or laboratory findings of Dependence, Abuse, intoxication, or withdrawal. Substance-Induced Sleep Disorder arises only in association with intoxication or withdrawal states, whereas the primary Sleep Disorders may precede the onset of substance use or occur during times of sustained abstinence. Because the withdrawal state for some substances with long half-lives (e.g., some benzodiazepines) can be relatively protracted, the onset of the sleep disturbance can occur up to 4 weeks after cessation of substance use but is usually seen within days of abstinence. Another consideration is the presence of features that are atypical of primary Sleep Disorders (e.g., atypical age at onset or course). In contrast, factors that suggest that the sleep disturbance is better accounted for by a primary Sleep Disorder include persistence of the sleep disturbance for more than about a month after the end of intoxication or acute withdrawal; the development of symptoms that are substantially in excess of what would be expected given the type or amount of the substance used or the duration of use; or a history of a prior primary Sleep Disorder.

Subtypes and Specifiers

The subtypes listed below can be used to indicate which of the following symptom presentations predominates. The clinical presentation of the specific Substance-Induced Sleep Disorder may resemble that of the analogous primary Sleep Disorder. However, the full criteria for the analogous primary Sleep Disorder do not need to be met to assign a diagnosis of Substance-Induced Sleep Disorder.

Insomnia Type. This subtype refers to a sleep complaint characterized primarily by difficulty falling asleep, difficulty maintaining sleep, or a feeling of nonrestorative sleep.

Hypersomnia Type. This subtype is used when the predominant complaint is one of excessively long nocturnal sleep or of excessive sleepiness during waking hours.

Parasomnia Type. This subtype refers to a sleep disturbance characterized primarily by abnormal behavioral events that occur in association with sleep or sleep-wake transitions.

Mixed Type. This subtype should be used to designate a substance-induced sleep problem characterized by multiple types of sleep symptoms but no symptom clearly predominates.

The context of the development of the sleep symptoms may be indicated by using one of the following specifiers:

With Onset During Intoxication. This specifier should be used if criteria are met for intoxication with the substance and symptoms develop during the intoxication syndrome.

With Onset During Withdrawal. This specifier should be used if criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, a withdrawal syndrome.

Recording Procedures

The name of the Substance-Induced Sleep Disorder begins with the specific substance (e.g., alcohol, methylphenidate, thyroxine) that is presumed to be causing the sleep disturbance. The diagnostic code is selected from the listing of classes of substances provided in the criteria set for Substance-Induced Sleep Disorder. For substances that do not fit into any of the classes (e.g., thyroxine), the code for "Other Substance" should be used. In addition, for medications prescribed at therapeutic doses, the specific medication can be indicated by listing the appropriate E-code (see Appendix G). The name of the disorder (e.g., Caffeine-Induced Sleep Disorder) is followed by the subtype indicating the predominant symptom presentation and the specifier indicating the context in which the symptoms developed (e.g., 292.89 Caffeine-Induced Sleep Disorder, Insomnia Type, With Onset During Intoxication). When more than one substance is judged to play a significant role in the development of the sleep disturbance, each should be listed separately (e.g., 292.89 Cocaine-Induced Sleep Disorder, Insomnia Type, With Onset During Intoxication; 291.89 Alcohol-Induced Sleep Disorder, Insomnia Type, With Onset During Withdrawal). If a substance is judged to be the etiological factor but the specific substance or class of substance is unknown, the category 292.89 Unknown Substance–Induced Sleep Disorder may be used.

Specific Substances

Substance-Induced Sleep Disorder most commonly occurs during **intoxication** with the following classes of substances: alcohol; amphetamine and related substances;

caffeine; cocaine; opioids; and sedatives, hypnotics, and anxiolytics. Sleep disturbances are also seen less commonly with intoxication with other types of substances. Substance-Induced Sleep Disorder can also occur in association with **withdrawal** from the following classes of substances: alcohol; amphetamine and related stimulants; cocaine; opioids; and sedatives, hypnotics, and anxiolytics. Each of the Substance-Induced Sleep Disorders produces EEG sleep patterns that are associated with, but cannot be considered diagnostic of, the disorder. The EEG sleep profile for each substance is further related to the stage of use, whether intoxication, chronic use, or withdrawal following discontinuation of the substance.

Alcohol. Alcohol-Induced Sleep Disorder typically occurs as the Insomnia Type. During acute intoxication, alcohol typically produces an immediate sedative effect, with increased sleepiness and reduced wakefulness for 3–4 hours. This is accompanied by an increase in stages 3 and 4 non–rapid eye movement (NREM) sleep and reduced rapid eye movement (REM) sleep during EEG sleep studies. Following these initial effects, the individual has increased wakefulness, restless sleep, and, often, vivid and anxiety-laden dreams for the rest of the sleep period. EEG sleep studies show that, in the second half of sleep after alcohol ingestion, stages 3 and 4 sleep is reduced, wakefulness is increased, and REM sleep is increased. Alcohol can aggravate Breathing-Related Sleep Disorder by increasing the number of obstructive apnea events. With continued habitual use, alcohol continues to show a short-lived sedative effect for several hours, followed by sleep continuity disruption for several hours.

During Alcohol Withdrawal, sleep is grossly disturbed. The individual typically has extremely disrupted sleep continuity, accompanied by an increase in the amount and intensity of REM sleep. This is often accompanied by an increase in vivid dreaming and, in the most extreme example, constitutes part of Alcohol Withdrawal Delirium. After acute withdrawal, individuals who have chronically used alcohol may continue to complain of light, fragmented sleep for weeks to years. EEG sleep studies confirm a persistent deficit in slow-wave sleep and persistent sleep continuity disturbance in these cases.

Amphetamines and related stimulants. Amphetamine-Induced Sleep Disorder is characterized by insomnia during intoxication and by hypersomnia during withdrawal. During the period of acute intoxication, amphetamine reduces the total amount of sleep, increases sleep latency and sleep continuity disturbances, increases body movements, and decreases REM sleep. Slow-wave sleep tends to be reduced. During withdrawal from chronic amphetamine use, individuals typically experience hypersomnia, with both prolonged nocturnal sleep duration and excessive sleepiness during the daytime. REM and slow-wave sleep may rebound to above baseline values. Multiple Sleep Latency Tests (MSLTs) may show increased daytime sleepiness during the withdrawal phase as well.

Caffeine. Caffeine-Induced Sleep Disorder typically produces insomnia, although some individuals may present with a complaint of hypersomnia and daytime sleepiness related to withdrawal (see p. 764). Caffeine exerts a dose-dependent effect, with increasing doses causing increased wakefulness and decreased sleep continuity. Polysomnography may show prolonged sleep latency, increased wakefulness, and

a decrease in slow-wave sleep. Consistent effects on REM sleep have not been described. Abrupt withdrawal from chronic caffeine use can produce hypersomnia. Some individuals may also experience hypersomnia between daytime doses of caffeine, as the immediate stimulant effect wanes.

Cocaine. As with other stimulants, cocaine typically produces insomnia during acute intoxication and hypersomnia during withdrawal. During acute intoxication, the total amount of sleep may be drastically reduced, with only short bouts of very disrupted sleep. Conversely, withdrawal after a cocaine binge is often associated with extremely prolonged sleep duration.

Opioids. During acute short-term use, opioids typically produce an increase in sleepiness and in subjective depth of sleep. REM sleep is typically reduced by acute administration of opioids, with little overall change in wakefulness or total sleep time. With continued administration, most individuals become tolerant to the sedative effects of opioids and may begin to complain of insomnia. This is mirrored by increased wakefulness and decreased sleep time in polysomnographic studies. Withdrawal from opioids is typically accompanied by hypersomnia complaints, although few objective studies have documented this finding.

Sedatives, hypnotics, and anxiolytics. Drugs within this class (e.g., barbiturates, benzodiazepines, meprobamate, glutethimide, and methyprylon) have similar, but not identical, effects on sleep. Differences in duration of action and half-life may affect sleep complaints and objective measures of sleep. In general, barbiturates and the older nonbarbiturate, nonbenzodiazepine drugs more consistently produce tolerance, dependence, and severe withdrawal, but these phenomena can be noted with benzodiazepines as well.

During acute intoxication, sedative-hypnotic drugs produce the expected increase in sleepiness and decrease in wakefulness. Polysomnographic studies confirm these subjective effects during acute administration, as well as a decrease in REM sleep and an increase in sleep-spindle activity. Chronic use (particularly of barbiturates and the older nonbarbiturate, nonbenzodiazepine drugs) may cause tolerance with the resulting return of insomnia. If the individual then increases the dose, daytime hypersomnia may occur. Sedative-hypnotic drugs can aggravate Breathing-Related Sleep Disorder by increasing the frequency and severity of obstructive sleep apnea events.

The abrupt discontinuation of chronic sedative-hypnotic use can lead to withdrawal insomnia. In addition to decreased sleep duration, withdrawal can produce increased anxiety, tremulousness, and ataxia. Barbiturates and the older nonbarbiturate, nonbenzodiazepine drugs are also associated with a high incidence of withdrawal seizures, which are much less frequently observed with benzodiazepines. Typically, sedative-hypnotic drugs with short durations of action are most likely to produce complaints of withdrawal insomnia, whereas those with longer durations of action are more often associated with daytime hypersomnia during active use. However, any sedative-hypnotic drug can potentially cause either daytime sedation or withdrawal insomnia. Withdrawal from sedative-hypnotic agents can be confirmed by polysomnographic studies, which show reduced sleep duration, increased sleep disruption, and REM sleep "rebound."

Other substances. Other substances may produce sleep disturbances. Common examples include medications that affect the central or autonomic nervous systems (including adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, and corticosteroids). Clinically, such medications are prescribed for the control of hypertension and cardiac arrhythmias, chronic obstructive pulmonary disease, gastrointestinal motility problems, or inflammatory processes.

Differential Diagnosis

Sleep disturbances are commonly encountered in the context of Substance Intoxication or Substance Withdrawal. A diagnosis of Substance-Induced Sleep Disorder should be made instead of a diagnosis of **Substance Intoxication** or **Substance Withdrawal** only when the sleep disturbance is judged to be in excess of that usually associated with the intoxication or withdrawal syndrome and when the disturbance is sufficiently severe to warrant independent clinical attention. For example, insomnia is a characteristic feature of Sedative, Hypnotic, or Anxiolytic Withdrawal. Sedative-, Hypnotic-, or Anxiolytic-Induced Sleep Disorder should be diagnosed instead of Sedative, Hypnotic, or Anxiolytic Withdrawal only if the insomnia is more severe than that usually encountered with Sedative, Hypnotic, or Anxiolytic Withdrawal and requires special attention and treatment. If the substance-induced sleep disturbance occurs exclusively during the course of a **delirium,** the sleep disturbance is considered to be an associated feature of the delirium and is not diagnosed separately. In **substance-induced presentations that contain a mix of different types of symptoms** (e.g., sleep, mood, and anxiety), the specific type of Substance-Induced Disorder to be diagnosed depends on which type of symptoms predominates in the clinical presentation.

A Substance-Induced Sleep Disorder is distinguished from a **primary Sleep Disorder** and from **Insomnia** or **Hypersomnia Related to Another Mental Disorder** by the fact that a substance is judged to be etiologically related to the symptoms (see p. 655).

A Substance-Induced Sleep Disorder due to a prescribed treatment for a mental disorder or general medical condition must have its onset while the person is receiving the medication (or during withdrawal, if there is a withdrawal syndrome associated with the medication). Once the treatment is discontinued, the sleep disturbance will usually remit within days to several weeks (depending on the half-life of the substance and the presence of a withdrawal syndrome). However, as discussed above, some form of sleep problem can persist at decreasing intensity for months following Sedative, Hypnotic, or Anxiolytic Withdrawal. With these exceptions, if symptoms persist beyond 4 weeks, other causes for the sleep disturbance should be considered. Not infrequently, individuals with a primary Sleep Disorder use medications or drugs of abuse to relieve their symptoms. If the clinician judges that the substance is playing a significant role in the exacerbation of the sleep disturbance, an additional diagnosis of a Substance-Induced Sleep Disorder may be warranted.

A Substance-Induced Sleep Disorder and **Sleep Disorder Due to a General Medical Condition** can also be difficult to distinguish. Both may produce similar symptoms of insomnia, hypersomnia, or (more rarely) a Parasomnia. Furthermore, many

individuals with general medical conditions that cause a sleep complaint are treated with medications that may also cause disturbances in sleep. The chronology of symptoms is the most important factor in distinguishing between these two causes of sleep disturbance. For instance, a sleep disturbance that clearly preceded the use of any medication for treatment of a general medical condition would suggest a diagnosis of Sleep Disorder Due to a General Medical Condition. Conversely, sleep symptoms that appear only after the institution of a particular medication or substance would suggest a Substance-Induced Sleep Disorder. In a similar way, a sleep disturbance that appears during treatment for a general medical condition but that improves after the medication is discontinued suggests a diagnosis of Substance-Induced Sleep Disorder. If the clinician has ascertained that the disturbance is due to both a general medical condition and substance use, both diagnoses (i.e., Sleep Disorder Due to a General Medical Condition and Substance-Induced Sleep Disorder) are given. When there is insufficient evidence to determine whether the sleep disturbance is due to a substance (including a medication) or to a general medical condition or is primary (i.e., not due to either a substance or a general medical condition), **Parasomnia Not Otherwise Specified** or **Dyssomnia Not Otherwise Specified** would be indicated.

Diagnostic criteria for Substance-Induced Sleep Disorder

A. A prominent disturbance in sleep that is sufficiently severe to warrant independent clinical attention.

B. There is evidence from the history, physical examination, or laboratory findings of either (1) or (2):

   (1) the symptoms in Criterion A developed during, or within a month of, Substance Intoxication or Withdrawal
   (2) medication use is etiologically related to the sleep disturbance

C. The disturbance is not better accounted for by a Sleep Disorder that is not substance induced. Evidence that the symptoms are better accounted for by a Sleep Disorder that is not substance induced might include the following: the symptoms precede the onset of the substance use (or medication use); the symptoms persist for a substantial period of time (e.g., about a month) after the cessation of acute withdrawal or severe intoxication or are substantially in excess of what would be expected given the type or amount of the substance used or the duration of use; or there is other evidence that suggests the existence of an independent non-substance-induced Sleep Disorder (e.g., a history of recurrent non-substance-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of Substance Intoxication or Substance Withdrawal only when the sleep symptoms are in excess of those usually associated with the intoxication or withdrawal syndrome and when the symptoms are sufficiently severe to warrant independent clinical attention.

Diagnostic criteria for Substance-Induced Sleep Disorder *(continued)*

Code [Specific Substance]–Induced Sleep Disorder:

(291.89 Alcohol; 292.89 Amphetamine; 292.89 Caffeine; 292.89 Cocaine; 292.89 Opioid; 292.89 Sedative, Hypnotic, or Anxiolytic; 292.89 Other [or Unknown] Substance)

Specify type:

Insomnia Type: if the predominant sleep disturbance is insomnia
Hypersomnia Type: if the predominant sleep disturbance is hypersomnia
Parasomnia Type: if the predominant sleep disturbance is a Parasomnia
Mixed Type: if more than one sleep disturbance is present and none predominates

Specify if (see table on p. 193 for applicability by substance):

With Onset During Intoxication: if the criteria are met for Intoxication with the substance and the symptoms develop during the intoxication syndrome
With Onset During Withdrawal: if criteria are met for Withdrawal from the substance and the symptoms develop during, or shortly after, a withdrawal syndrome

Impulse-Control Disorders Not Elsewhere Classified

This section includes disorders of impulse control that are not classified as part of the presentation of disorders in other sections of the manual (e.g., Substance-Related Disorders, Paraphilias, Antisocial Personality Disorder, Conduct Disorder, Schizophrenia, and Mood Disorders may have features that involve problems of impulse control). The essential feature of Impulse-Control Disorders is the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others. For most of the disorders in this section, the individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing the act. Following the act there may or may not be regret, self-reproach, or guilt. The following disorders are included in this section:

Intermittent Explosive Disorder is characterized by discrete episodes of failure to resist aggressive impulses resulting in serious assaults or destruction of property.

Kleptomania is characterized by the recurrent failure to resist impulses to steal objects not needed for personal use or monetary value.

Pyromania is characterized by a pattern of fire setting for pleasure, gratification, or relief of tension.

Pathological Gambling is characterized by recurrent and persistent maladaptive gambling behavior.

Trichotillomania is characterized by recurrent pulling out of one's hair for pleasure, gratification, or relief of tension that results in noticeable hair loss.

Impulse-Control Disorder Not Otherwise Specified is included for coding disorders of impulse control that do not meet the criteria for any of the specific Impulse-Control Disorders described above or in other sections of the manual.

312.34 Intermittent Explosive Disorder

Diagnostic Features

The essential feature of Intermittent Explosive Disorder is the occurrence of discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property (Criterion A). Examples of serious assaultive acts include striking or otherwise hurting another person or verbally threatening to physically assault another individual. Destruction of property entails purposeful breaking of an object of value; minor or unintentional damage is not of sufficient severity to meet this criterion. The degree of aggressiveness expressed during an episode is grossly

out of proportion to any provocation or precipitating psychosocial stressor (Criterion B). A diagnosis of Intermittent Explosive Disorder is made only after other mental disorders that might account for episodes of aggressive behavior have been ruled out (e.g., Antisocial Personality Disorder, Borderline Personality Disorder, a Psychotic Disorder, a Manic Episode, Conduct Disorder, or Attention-Deficit/Hyperactivity Disorder) (Criterion C). The aggressive episodes are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., head trauma, Alzheimer's disease) (Criterion C). The individual may describe the aggressive episodes as "spells" or "attacks" in which the explosive behavior is preceded by a sense of tension or arousal and is followed immediately by a sense of relief. Later the individual may feel upset, remorseful, regretful, or embarrassed about the aggressive behavior.

Associated Features and Disorders

Associated descriptive features and mental disorders. Individuals with Intermittent Explosive Disorder sometimes describe intense impulses to be aggressive prior to their aggressive acts. Explosive episodes may be associated with affective symptoms (irritability or rage, increased energy, racing thoughts) during the aggressive impulses and acts, and rapid onset of depressed mood and fatigue after the acts. Some individuals may also report that their aggressive episodes are often preceded or accompanied by symptoms such as tingling, tremor, palpitations, chest tightness, head pressure, or hearing an echo. Individuals may describe their aggressive impulses as extremely distressing. The disorder may result in job loss, school suspension, divorce, difficulties with interpersonal relationships or other impairment in social or occupational spheres, accidents (e.g., in vehicles), hospitalization (e.g., because of injuries incurred in fights or accidents), financial problems, incarcerations, or other legal problems.

Signs of generalized impulsivity or aggressiveness may be present between explosive episodes. Individuals with Intermittent Explosive Disorder may report problems with chronic anger and frequent "subthreshold" episodes, in which they experience aggressive impulses but either manage to resist acting on them or engage in less destructive aggressive behaviors (e.g., screaming, punching a wall without damaging it).

Individuals with narcissistic, obsessive, paranoid, or schizoid traits may be especially prone to having explosive outbursts of anger when under stress. Preliminary data suggest that Mood Disorders, Anxiety Disorders, Eating Disorders, Substance Use Disorders, and other Impulse-Control Disorders may be associated with Intermittent Explosive Disorder. Childhood histories may show severe temper tantrums, impaired attention, hyperactivity, and other behavioral difficulties, such as stealing and fire setting.

Associated laboratory findings. There may be nonspecific EEG findings (e.g., slowing) or evidence of abnormalities on neuropsychological testing (e.g., difficulty with letter reversal). Signs of altered serotonin metabolism (e.g., low mean 5-hydroxyindoleacetic acid [5-HIAA] concentrations) have been found in the cerebrospinal fluid of some impulsive and temper-prone individuals, but the specific relationship of these findings to Intermittent Explosive Disorder is unclear.

Associated physical examination findings and general medical conditions. There may be nonspecific or "soft" findings on neurological examinations (e.g., reflex asymmetries or mirror movements). Developmental difficulties indicative of cerebral dysfunction may be present (e.g., delayed speech or poor coordination). A history of neurological conditions (e.g., migraine headaches, head injury, episodes of unconsciousness, or febrile seizures in childhood) may be present. However, if the clinician judges that the aggressive behavior is a consequence of the direct physiological effects of a diagnosable general medical condition, the appropriate Mental Disorder Due to a General Medical Condition should be diagnosed instead (e.g., Personality Change Due to Head Trauma, Aggressive Type; Dementia of the Alzheimer's Type, Early Onset, Uncomplicated, With Behavioral Disturbance).

Specific Culture and Gender Features

Amok is characterized by an episode of acute, unrestrained violent behavior for which the person claims amnesia. Although traditionally seen in southeastern Asian countries, cases of amok have been reported in Canada and the United States. Unlike Intermittent Explosive Disorder, amok typically occurs as a single episode rather than as a pattern of aggressive behavior and is often associated with prominent dissociative features. Episodic violent behavior is more common in males than in females.

Prevalence

Reliable information is lacking, but Intermittent Explosive Disorder is apparently rare.

Course

Limited data are available on the age at onset of Intermittent Explosive Disorder, but it appears to be from childhood to the early 20s. Mode of onset may be abrupt and without a prodromal period. The course of Intermittent Explosive Disorder is variable, with the disorder having a chronic course in some individuals and a more episodic course in other individuals.

Familial Pattern

Mood Disorders, Substance Use Disorders, Intermittent Explosive Disorder, and other Impulse-Control Disorders may be more common among the first-degree relatives of individuals with Intermittent Explosive Disorder than among the general population.

Differential Diagnosis

Aggressive behavior can occur in the context of many other mental disorders. A diagnosis of Intermittent Explosive Disorder should be considered only after all other disorders that are associated with aggressive impulses or behavior have been ruled out. If the aggressive behavior occurs exclusively during the course of a **delirium,** a

diagnosis of Intermittent Explosive Disorder is not given. Similarly, when the behavior develops as part of a **dementia,** a diagnosis of Intermittent Explosive Disorder is not made and the appropriate diagnosis is dementia with the specifier With Behavioral Disturbance. Intermittent Explosive Disorder should be distinguished from **Personality Change Due to a General Medical Condition, Aggressive Type,** which is diagnosed when the pattern of aggressive episodes is judged to be due to the direct physiological effects of a diagnosable general medical condition (e.g., an individual who has suffered brain injury from an automobile accident and subsequently manifests a change in personality characterized by aggressive outbursts). In rare cases, episodic violence may occur in individuals with epilepsy, especially of frontal and temporal origin (partial complex epilepsy).

A careful history and a thorough neurological evaluation are helpful in making the determination. Note that nonspecific abnormalities on neurological examination (e.g., "soft signs") and nonspecific EEG changes are compatible with a diagnosis of Intermittent Explosive Disorder and only preempt the diagnosis if they are indicative of a diagnosable general medical condition.

Aggressive outbursts may also occur in association with **Substance Intoxication** or **Substance Withdrawal,** particularly associated with alcohol, phencyclidine, cocaine and other stimulants, barbiturates, and inhalants. The clinician should inquire carefully about the nature and extent of substance use, and a blood or urine drug screen may be informative.

Intermittent Explosive Disorder should be distinguished from the aggressive or erratic behavior that can occur in **Oppositional Defiant Disorder, Conduct Disorder, Antisocial Personality Disorder, Borderline Personality Disorder,** a **Manic Episode,** and **Schizophrenia.** If the aggressive behavior is better accounted for as a diagnostic or associated feature of another mental disorder, a separate diagnosis of Intermittent Explosive Disorder is not given. However, impulsive aggression in individuals with Antisocial Personality Disorder and Borderline Personality Disorder can have specific clinical relevance, in which case both diagnoses may be made. For example, if an individual with an established diagnosis of Borderline Personality Disorder develops discrete episodes of failure to resist aggressive impulses resulting in serious physical or verbal assaultive acts or destruction of property, an additional diagnosis of Intermittent Explosive Disorder may be warranted.

"Anger attacks"—sudden spells of anger associated with autonomic arousal (tachycardia, sweating, flushing) and feelings of being out of control—have been described in individuals with **Major Depressive Disorder** and **Panic Disorder.** If these attacks occur only in the setting of a Major Depressive Episode or a Panic Attack, they should not count toward a diagnosis of Intermittent Explosive Disorder. However, if these anger attacks also occur at times other than during Major Depressive Episodes or Panic Attacks, and meet the Intermittent Explosive Disorder criterion for serious assaultive acts, then both diagnoses may be given.

Aggressive behavior may, of course, occur when no mental disorder is present. **Purposeful behavior** is distinguished from Intermittent Explosive Disorder by the presence of motivation and gain in the aggressive act. In forensic settings, individuals may **malinger** Intermittent Explosive Disorder to avoid responsibility for their behavior. **Anger as a normal reaction to specific life events or environmental situations** also needs to be distinguished from the anger that may occur as part of an

aggressive episode in Intermittent Explosive Disorder, which occurs with little or no provocation.

Diagnostic criteria for 312.34 Intermittent Explosive Disorder

A. Several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property.

B. The degree of aggressiveness expressed during the episodes is grossly out of proportion to any precipitating psychosocial stressors.

C. The aggressive episodes are not better accounted for by another mental disorder (e.g., Antisocial Personality Disorder, Borderline Personality Disorder, a Psychotic Disorder, a Manic Episode, Conduct Disorder, or Attention-Deficit/Hyperactivity Disorder) and are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., head trauma, Alzheimer's disease).

312.32 Kleptomania

Diagnostic Features

The essential feature of Kleptomania is the recurrent failure to resist impulses to steal items even though the items are not needed for personal use or for their monetary value (Criterion A). The individual experiences a rising subjective sense of tension before the theft (Criterion B) and feels pleasure, gratification, or relief when committing the theft (Criterion C). The stealing is not committed to express anger or vengeance, is not done in response to a delusion or hallucination (Criterion D), and is not better accounted for by Conduct Disorder, a Manic Episode, or Antisocial Personality Disorder (Criterion E). The objects are stolen despite the fact that they are typically of little value to the individual, who could have afforded to pay for them and often gives them away or discards them. Occasionally the individual may hoard the stolen objects or surreptitiously return them. Although individuals with this disorder will generally avoid stealing when immediate arrest is probable (e.g., in full view of a police officer), they usually do not preplan the thefts or fully take into account the chances of apprehension. The stealing is done without assistance from, or collaboration with, others.

Associated Features and Disorders

Individuals with Kleptomania experience the impulse to steal as ego-dystonic and are aware that the act is wrong and senseless. The person frequently fears being apprehended and often feels depressed or guilty about the thefts. Kleptomania may be associated with compulsive buying as well as with Mood Disorders (especially Major Depressive Disorder), Anxiety Disorders, Eating Disorders (particularly Bulimia

Nervosa), Personality Disorders, and other Impulse-Control Disorders. The disorder may cause legal, family, career, and personal difficulties.

Specific Gender Features

Preliminary evidence suggests that, in clinical samples, approximately two-thirds of individuals with Kleptomania are female.

Prevalence

Kleptomania is a rare condition that appears to occur in fewer than 5% of identified shoplifters. Its prevalence in the general population is unknown.

Course

Age at onset of Kleptomania is variable. The disorder may begin in childhood, adolescence, or adulthood, and in rare cases in late adulthood. There is little systematic information on the course of Kleptomania, but three typical courses have been described: sporadic with brief episodes and long periods of remission; episodic with protracted periods of stealing and periods of remission; and chronic with some degree of fluctuation. The disorder may continue for years, despite multiple convictions for shoplifting.

Familial Pattern

There are no controlled family history studies of Kleptomania. However, preliminary data suggest that first-degree relatives of individuals with Kleptomania may have higher rates of Obsessive-Compulsive Disorder than the general population.

Differential Diagnosis

Kleptomania should be distinguished from **ordinary acts of theft or shoplifting.** Ordinary theft (whether planned or impulsive) is deliberate and is motivated by the usefulness of the object or its monetary worth. Some individuals, especially adolescents, may also steal on a dare, as an act of rebellion, or as a rite of passage. The diagnosis is not made unless other characteristic features of Kleptomania are also present. Kleptomania is exceedingly rare, whereas shoplifting is relatively common. In **Malingering,** individuals may simulate the symptoms of Kleptomania to avoid criminal prosecution. **Antisocial Personality Disorder** and **Conduct Disorder** are distinguished from Kleptomania by a general pattern of antisocial behavior. Kleptomania should be distinguished from intentional or inadvertent stealing that may occur during a **Manic Episode,** in response to delusions or hallucinations (e.g., in **Schizophrenia**), or as a result of a **dementia.**

Diagnostic criteria for 312.32 Kleptomania

A. Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value.

B. Increasing sense of tension immediately before committing the theft.

C. Pleasure, gratification, or relief at the time of committing the theft.

D. The stealing is not committed to express anger or vengeance and is not in response to a delusion or a hallucination.

E. The stealing is not better accounted for by Conduct Disorder, a Manic Episode, or Antisocial Personality Disorder.

312.33 Pyromania

Diagnostic Features

The essential feature of Pyromania is the presence of multiple episodes of deliberate and purposeful fire setting (Criterion A). Individuals with this disorder experience tension or affective arousal before setting a fire (Criterion B). There is a fascination with, interest in, curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences) (Criterion C). Individuals with this disorder are often regular "watchers" at fires in their neighborhoods, may set off false alarms, and derive pleasure from institutions, equipment, and personnel associated with fire. They may spend time at the local fire department, set fires to be affiliated with the fire department, or even become firefighters. Individuals with this disorder experience pleasure, gratification, or a release of tension when setting the fire, witnessing its effects, or participating in its aftermath (Criterion D). The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's living circumstances, or in response to a delusion or a hallucination (Criterion E). The fire setting does not result from impaired judgment (e.g., in dementia or Mental Retardation). The diagnosis is not made if the fire setting is better accounted for by Conduct Disorder, a Manic Episode, or Antisocial Personality Disorder (Criterion F).

Associated Features and Disorders

Individuals with Pyromania may make considerable advance preparation for starting a fire. They may be indifferent to the consequences to life or property caused by the fire, or they may derive satisfaction from the resulting property destruction. The behaviors may lead to property damage, legal consequences, or injury or loss of life to the fire setter or to others. Individuals who impulsively set fires (who may or may not have Pyromania) often have a current or past history of Alcohol Dependence or Abuse.

Specific Age and Gender Features

Although fire setting is a major problem in children and adolescents (over 40% of those arrested for arson offenses in the United States are under age 18 years), Pyromania in childhood appears to be rare. Juvenile fire setting is usually associated with Conduct Disorder, Attention-Deficit/Hyperactivity Disorder, or Adjustment Disorder. Pyromania occurs much more often in males, especially those with poorer social skills and learning difficulties.

Prevalence

Pyromania is apparently rare.

Course

There are insufficient data to establish a typical age at onset of Pyromania. The relationship between fire setting in childhood and Pyromania in adulthood has not been documented. In individuals with Pyromania, fire-setting incidents are episodic and may wax and wane in frequency. Longitudinal course is unknown.

Differential Diagnosis

It is important to rule out other causes of fire setting before giving the diagnosis of Pyromania. Intentional fire setting may occur for **profit, sabotage, or revenge; to conceal a crime; to make a political statement** (e.g., an act of terrorism or protest); or **to attract attention or recognition** (e.g., setting a fire in order to discover it and save the day). Fire setting may also occur as part of **developmental experimentation in childhood** (e.g., playing with matches, lighters, or fire). Some individuals with mental disorders use fire setting to communicate a desire, wish, or need, often directed at gaining a change in the nature or location of services. This form of fire setting has been referred to as "communicative arson" and must be carefully distinguished from Pyromania. A separate diagnosis of Pyromania is not given when fire setting occurs as part of **Conduct Disorder,** a **Manic Episode,** or **Antisocial Personality Disorder,** or if it occurs in response to a delusion or a hallucination (e.g., in **Schizophrenia**) or if it is due to the direct physiological effects of a general medical condition (e.g., epilepsy). The diagnosis of Pyromania should also not be given when fire setting results from impaired judgment associated with **dementia, Mental Retardation,** or **Substance Intoxication.**

Diagnostic criteria for 312.33 Pyromania

A. Deliberate and purposeful fire setting on more than one occasion.

B. Tension or affective arousal before the act.

C. Fascination with, interest in, curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences).

D. Pleasure, gratification, or relief when setting fires, or when witnessing or participating in their aftermath.

E. The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's living circumstances, in response to a delusion or hallucination, or as a result of impaired judgment (e.g., in dementia, Mental Retardation, Substance Intoxication).

F. The fire setting is not better accounted for by Conduct Disorder, a Manic Episode, or Antisocial Personality Disorder.

312.31 Pathological Gambling

Diagnostic Features

The essential feature of Pathological Gambling is persistent and recurrent maladaptive gambling behavior (Criterion A) that disrupts personal, family, or vocational pursuits. The diagnosis is not made if the gambling behavior is better accounted for by a Manic Episode (Criterion B).

The individual may be preoccupied with gambling (e.g., reliving past gambling experiences, planning the next gambling venture, or thinking of ways to get money with which to gamble) (Criterion A1). Most individuals with Pathological Gambling say that they are seeking "action" (an aroused, euphoric state) or excitement even more than money. Increasingly larger bets, or greater risks, may be needed to continue to produce the desired level of excitement (Criterion A2). Individuals with Pathological Gambling often continue to gamble despite repeated efforts to control, cut back, or stop the behavior (Criterion A3). There may be restlessness or irritability when attempting to cut down or stop gambling (Criterion A4). The individual may gamble as a way of escaping from problems or to relieve a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression) (Criterion A5). A pattern of "chasing" one's losses may develop, with an urgent need to keep gambling (often with larger bets or the taking of greater risks) to undo a loss or series of losses. The individual may abandon his or her gambling strategy and try to win back losses all at once. Although all gamblers may chase for short periods, it is the long-term chase that is more characteristic of individuals with Pathological Gambling (Criterion A6). The individual may lie to family members, therapists, or others to conceal the extent of involvement with gambling (Criterion A7). When the individual's borrowing resources are

strained, the person may resort to antisocial behavior (e.g., forgery, fraud, theft, or embezzlement) to obtain money (Criterion A8). The individual may have jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling (Criterion A9). The individual may also engage in "bailout" behavior, turning to family or others for help with a desperate financial situation that was caused by gambling (Criterion A10).

Associated Features and Disorders

Associated descriptive features and mental disorders. Distortions in thinking (e.g., denial, superstitions, overconfidence, or a sense of power and control) may be present in individuals with Pathological Gambling. Many individuals with Pathological Gambling believe that money is both the cause of and solution to all their problems. Individuals with Pathological Gambling are frequently highly competitive, energetic, restless, and easily bored. They may be overly concerned with the approval of others and may be generous to the point of extravagance. When not gambling, they may be workaholics or "binge" workers who wait until they are up against deadlines before really working hard. They may be prone to developing general medical conditions that are associated with stress (e.g., hypertension, peptic ulcer disease, migraine). Individuals seeking treatment for Pathological Gambling have relatively high rates of suicidal ideation and suicide attempts. Studies of men with Pathological Gambling suggest that a history of inattentive and hyperactive symptoms in childhood may be a risk factor for development of Pathological Gambling later in life. Increased rates of Mood Disorders, Attention-Deficit/Hyperactivity Disorder, Substance Abuse or Dependence, other Impulse-Control Disorders, and Antisocial, Narcissistic, and Borderline Personality Disorders have been reported in individuals with Pathological Gambling.

Associated laboratory findings. There are no laboratory findings that are diagnostic of Pathological Gambling. However, a variety of laboratory findings have been reported to be abnormal in males with Pathological Gambling compared with control subjects. These include measures of neurotransmitters and their metabolites in cerebrospinal fluid and urine, and response to neuroendocrine challenges, implicating abnormalities in a variety of neurotransmitter systems, including the serotonin, norepinephrine, and dopamine systems. Abnormalities in platelet monoamine oxidase activity have also been reported in males with Pathological Gambling. Individuals with Pathological Gambling may display high levels of impulsivity on neuropsychological tests.

Specific Culture and Gender Features

There are cultural variations in the prevalence and type of gambling activities (e.g., pai go, cockfights, horse racing, the stock market). Approximately one-third of individuals with Pathological Gambling are females, but in different geographic areas and cultures, gender ratio can vary considerably. Females with the disorder are more apt to be depressed and to gamble as an escape. Females are underrepresented in treatment programs for gambling and represent only 2%–4% of the population of

Gamblers Anonymous. This may be a function of the greater stigma attached to female gamblers.

Prevalence

The prevalence of Pathological Gambling is influenced by both the availability of gambling and the duration of availability such that with the increasing availability of legalized gambling, there is an increase in the prevalence of Pathological Gambling. Community studies estimate the lifetime prevalence of Pathological Gambling to range from 0.4% to 3.4% in adults, although prevalence rates in some areas (e.g., Puerto Rico, Australia) have been reported to be as high as 7%. Higher prevalence rates, ranging from 2.8% to 8%, have been reported in adolescents and college students. The prevalence of Pathological Gambling may be increased in treatment-seeking individuals with a Substance Use Disorder.

Course

Pathological Gambling typically begins in early adolescence in males and later in life in females. Although a few individuals are "hooked" with their very first bet, for most the course is more insidious. There may be years of social gambling followed by an abrupt onset that may be precipitated by greater exposure to gambling or by a stressor. The gambling pattern may be regular or episodic, and the course of the disorder is typically chronic. There is generally a progression in the frequency of gambling, the amount wagered, and the preoccupation with gambling and obtaining money with which to gamble. The urge to gamble and gambling activity generally increase during periods of stress or depression.

Familial Pattern

Pathological Gambling and Alcohol Dependence are both more common among the parents of individuals with Pathological Gambling than among the general population.

Differential Diagnosis

Pathological Gambling must be distinguished from social gambling and professional gambling. **Social gambling** typically occurs with friends or colleagues and lasts for a limited period of time, with predetermined acceptable losses. In **professional gambling,** risks are limited and discipline is central. Some individuals can experience problems associated with their gambling (e.g., short-term chasing behavior and loss of control) that do not meet the full criteria for Pathological Gambling.

Loss of judgment and excessive gambling may occur during a **Manic Episode.** An additional diagnosis of Pathological Gambling should only be given if the gambling behavior is not better accounted for by the Manic Episode (e.g., a history of maladaptive gambling behavior at times other than during a Manic Episode). Alternatively, an individual with Pathological Gambling may exhibit behavior during a gambling binge that resembles a Manic Episode. However, once the individual is away from the

gambling, these manic-like features dissipate. Problems with gambling may occur in individuals with **Antisocial Personality Disorder**; if criteria are met for both disorders, both can be diagnosed.

Diagnostic criteria for 312.31 Pathological Gambling

A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:

(1) is preoccupied with gambling (e.g., preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble)
(2) needs to gamble with increasing amounts of money in order to achieve the desired excitement
(3) has repeated unsuccessful efforts to control, cut back, or stop gambling
(4) is restless or irritable when attempting to cut down or stop gambling
(5) gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)
(6) after losing money gambling, often returns another day to get even ("chasing" one's losses)
(7) lies to family members, therapist, or others to conceal the extent of involvement with gambling
(8) has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling
(9) has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling
(10) relies on others to provide money to relieve a desperate financial situation caused by gambling

B. The gambling behavior is not better accounted for by a Manic Episode.

312.39 Trichotillomania

Diagnostic Features

The essential feature of Trichotillomania is the recurrent pulling out of one's own hair that results in noticeable hair loss (Criterion A). Sites of hair pulling may include any region of the body in which hair may grow (including axillary, pubic, and perirectal regions), with the most common sites being the scalp, eyebrows, and eyelashes. Hair pulling may occur in brief episodes scattered throughout the day or in less frequent but more sustained periods that can continue for hours. Hair pulling often occurs in states of relaxation and distraction (e.g., when reading a book or watching television) but may also occur during stressful circumstances. An increasing sense of tension is present immediately before pulling out the hair (Criterion B). For some, tension does not necessarily precede the act but is associated with attempts to resist the urge. There is gratification, pleasure, or a sense of relief when pulling out the hair (Criterion C). Some

individuals experience an "itchlike" sensation in the scalp that is eased by the act of pulling hair. The diagnosis is not given if the hair pulling is better accounted for by another mental disorder (e.g., in response to a delusion or a hallucination) or is due to a general medical condition (e.g., inflammation of the skin or other dermatological conditions) (Criterion D). The disturbance must cause significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E).

Associated Features and Disorders

Associated descriptive features and mental disorders. Examining the hair root, twirling it off, pulling the strand between the teeth, or trichophagia (eating hairs) may occur with Trichotillomania. Hair pulling does not usually occur in the presence of other people (except immediate family members), and social situations may be avoided. Individuals commonly deny their hair-pulling behavior and conceal or camouflage the resulting alopecia. Some individuals have urges to pull hairs from other people and may sometimes try to find opportunities to do so surreptitiously. They may pull hairs from pets, dolls, and other fibrous materials (e.g., sweaters or carpets). Nail biting, scratching, gnawing, and excoriation is often associated with Trichotillomania. Individuals with Trichotillomania may also have Mood Disorders, Anxiety Disorders (especially Obsessive-Compulsive Disorder), Substance Use Disorders, Eating Disorders, Personality Disorders, or Mental Retardation.

Associated laboratory findings. Certain histological findings are considered characteristic and may aid diagnosis when Trichotillomania is suspected and the affected individual denies symptoms. Biopsy samples from involved areas may reveal short and broken hairs. Histological examination will reveal normal and damaged follicles in the same area, as well as an increased number of catagen hairs. Some hair follicles may show signs of trauma (wrinkling of the outer root sheath). Involved follicles may be empty or may contain a deeply pigmented keratinous material. The absence of inflammation distinguishes Trichotillomania-induced alopecia from alopecia areata.

Associated physical examination findings and general medical conditions. Pain is not routinely reported to accompany the hair pulling; pruritus and tingling in the involved areas may be present. The patterns of hair loss are highly variable. Areas of complete alopecia are common, as well as areas of noticeably thinned hair density. When the scalp is involved, there may be a predilection for the crown or parietal regions. The surface of the scalp usually shows no evidence of excoriation. There may be a pattern of nearly complete baldness except for a narrow perimeter around the outer margins of the scalp, particularly at the nape of the neck ("tonsure trichotillomania"). Eyebrows and eyelashes may be completely absent. Thinning of pubic hairs may be apparent on inspection. There may be areas of absent hair on the limbs or torso. Trichophagia may result in bezoars (hair balls) that may lead to anemia, abdominal pain, hematemesis, nausea and vomiting, and bowel obstruction and even perforation.

Specific Culture, Age, and Gender Features

Among children with Trichotillomania, males and females are equally represented. Among adults, Trichotillomania is much more common among females than among males. This may reflect the true gender ratio of the condition, or it may reflect differential treatment seeking based on cultural or gender-based attitudes regarding appearance (e.g., acceptance of normative hair loss among males).

Prevalence

No systematic data are available on the prevalence of Trichotillomania. Although Trichotillomania was previously thought to be an uncommon condition, it is now believed to occur more frequently. For example, a survey of college students found a lifetime rate of 0.6%.

Course

Transient periods of hair pulling in early childhood may be considered a benigı "habit" with a self-limited course. Individuals who present with chronic Trichotillomania in adulthood often report onset in early adolescence. Some individuals have continuous symptoms for decades. For others, the disorder may come and go for weeks, months, or years at a time. Sites of hair pulling may vary over time.

Differential Diagnosis

Other causes of alopecia should be considered in individuals who deny hair pulling (e.g., alopecia areata, male-pattern baldness, chronic discoid lupus erythematosus, lichen planopilaris, folliculitis decalvans, pseudopelade, and alopecia mucinosa). A separate diagnosis of Trichotillomania is not given if the behavior is better accounted for by **another mental disorder** (e.g., in response to a delusion or a hallucination in Schizophrenia). The repetitive hair pulling in Trichotillomania must be distinguished from a compulsion, as in **Obsessive-Compulsive Disorder.** In Obsessive-Compulsive Disorder, the repetitive behaviors are performed in response to an obsession, or according to rules that must be applied rigidly. An additional diagnosis of **Stereotypic Movement Disorder** is not made if the repetitive behavior is limited t hair pulling. The self-induced alopecia in Trichotillomania must be distinguishe from **Factitious Disorder With Predominantly Physical Signs and Symptoms,** in which the motivation for the behavior is assuming the sick role.

Many individuals twist and play with hair, especially during states of heightened anxiety, but this behavior does not usually qualify for a diagnosis of Trichotillomania. Some individuals may present with features of Trichotillomania, but the resulting hair damage may be so slight as to be virtually undetectable. In such situations, the diagnosis should only be considered if the individual experiences significant distress. In children, self-limited periods of hair pulling are common and may be considered a temporary "habit." This form of childhood hair pulling differs from adult forms of Trichotillomania in that there may be an absence of reported tension or relief associated with the hair pulling. Therefore, among children, the diagnosis should be reserved for situations in which the behavior has persisted for several months.

Diagnostic criteria for 312.39 Trichotillomania

A. Recurrent pulling out of one's hair resulting in noticeable hair loss.

B. An increasing sense of tension immediately before pulling out the hair or when attempting to resist the behavior.

C. Pleasure, gratification, or relief when pulling out the hair.

D. The disturbance is not better accounted for by another mental disorder and is not due to a general medical condition (e.g., a dermatological condition).

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

312.30 Impulse-Control Disorder Not Otherwise Specified

This category is for disorders of impulse control (e.g., skin picking) that do not meet the criteria for any specific Impulse-Control Disorder or for another mental disorder having features involving impulse control described elsewhere in the manual (e.g., Substance Dependence, a Paraphilia).

Adjustment Disorders

Diagnostic Features

The essential feature of an Adjustment Disorder is a psychological response to an identifiable stressor or stressors that results in the development of clinically significant emotional or behavioral symptoms. The symptoms must develop within 3 months after the onset of the stressor(s) (Criterion A). The clinical significance of the reaction is indicated either by marked distress that is in excess of what would be expected given the nature of the stressor or by significant impairment in social or occupational (academic) functioning (Criterion B). In other words, a reaction to a stressor that might be considered normal or expectable can still qualify for a diagnosis of Adjustment Disorder if the reaction is sufficiently severe to cause significant impairment. This category should not be used if the disturbance meets the criteria for another specific Axis I disorder (e.g., a specific Anxiety or Mood Disorder) or is merely an exacerbation of a preexisting Axis I or II disorder (Criterion C). However, an Adjustment Disorder may be diagnosed in the presence of another Axis I or Axis II disorder if the latter does not account for the pattern of symptoms that have occurred in response to the stressor. The diagnosis of an Adjustment Disorder also does not apply when the symptoms represent Bereavement (Criterion D). By definition, an Adjustment Disorder must resolve within 6 months of the termination of the stressor (or its consequences) (Criterion E). However, the symptoms may persist for a prolonged period (i.e., longer than 6 months) if they occur in response to a chronic stressor (e.g., a chronic, disabling general medical condition) or to a stressor that has enduring consequences (e.g., the financial and emotional difficulties resulting from a divorce).

The stressor may be a single event (e.g., termination of a romantic relationship), or there may be multiple stressors (e.g., marked business difficulties and marital problems). Stressors may be recurrent (e.g., associated with seasonal business crises) or continuous (e.g., living in a crime-ridden neighborhood). Stressors may affect a single individual, an entire family, or a larger group or community (e.g., as in a natural disaster). Some stressors may accompany specific developmental events (e.g., going to school, leaving the parental home, getting married, becoming a parent, failing to attain occupational goals, retirement).

Subtypes and Specifiers

Adjustment Disorders are coded according to the subtype that best characterizes the predominant symptoms:

> **309.0 With Depressed Mood.** This subtype should be used when the predominant manifestations are symptoms such as depressed mood, tearfulness, or feelings of hopelessness.

309.24 With Anxiety. This subtype should be used when the predominant manifestations are symptoms such as nervousness, worry, or jitteriness, or, in children, fears of separation from major attachment figures.

309.28 With Mixed Anxiety and Depressed Mood. This subtype should be used when the predominant manifestation is a combination of depression and anxiety.

309.3 With Disturbance of Conduct. This subtype should be used when the predominant manifestation is a disturbance in conduct in which there is violation of the rights of others or of major age-appropriate societal norms and rules (e.g., truancy, vandalism, reckless driving, fighting, defaulting on legal responsibilities).

309.4 With Mixed Disturbance of Emotions and Conduct. This subtype should be used when the predominant manifestations are both emotional symptoms (e.g., depression, anxiety) and a disturbance of conduct (see above subtype).

309.9 Unspecified. This subtype should be used for maladaptive reactions (e.g., physical complaints, social withdrawal, or work or academic inhibition) to stressors that are not classifiable as one of the specific subtypes of Adjustment Disorder.

The duration of the symptoms of an Adjustment Disorder can be indicated by choosing one of the following specifiers:

Acute. This specifier can be used to indicate persistence of symptoms for less than 6 months.

Chronic. This specifier can be used to indicate persistence of symptoms for 6 months or longer. By definition, symptoms cannot persist for more than 6 months after the termination of the stressor or its consequences. The Chronic specifier therefore applies when the duration of the disturbance is longer than 6 months in response to a chronic stressor or to a stressor that has enduring consequences.

Recording Procedures

The predominant symptom presentation for an Adjustment Disorder should be indicated by choosing the diagnostic code and term from the list above, followed, if desired, by the Acute or Chronic specifier (e.g., 309.0 Adjustment Disorder With Depressed Mood, Acute). In a multiaxial assessment, the nature of the stressor can be indicated by listing it on Axis IV (e.g., Divorce).

Associated Features and Disorders

The subjective distress or impairment in functioning associated with Adjustment Disorders is frequently manifested as decreased performance at work or school and temporary changes in social relationships. Adjustment Disorders are associated with suicide attempts, suicide, excessive substance use, and somatic complaints. Adjustment Disorder has been reported in individuals with preexisting mental disorders in selected samples, such as children and adolescents and in general medical and surgi-